# HEART DISEASE IN PREGNANCY.

By

DAVID CAMERON HAIG,
M.B., Ch.B., M.R.C.P. Ed.

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## INTRODUCTION.

The association of pregnancy and heart disease has until recent years been regarded with apprehension by obstetricians and physicians alike and has in the past been responsible for countless therapeutic abortions by otherwise conservative physicians. Recent experience has in general led to a more optimistic view but there is still a fairly wide divergence of opinion among competent observers concerning the ability of the cardiac patient to bear children and there is an even wider divergence of opinion as to the ultimate effects of childbearing on the expected duration of life of cardiac patients.

Sir William Osler once stated: "It is of use from time to time, to take stock, so to speak, of our knowledge of a particular disease, to see exactly where we stand with regard to it, to inquire to what conclusion the accummulated facts seem to point and to ascertain in what direction we may look for fruitful investigation in the future." An attempt has been made in this thesis to survey the problem of heart disease in association with pregnancy both in its immediate effects and its ultimate effects.

/Material.....

Material for this study has been made available by the fact that since 1928 a well-conducted Cardiac Clinic has been attached to the Ante-Natal Department of the Royal Simpson Maternity Pavilion (formerly the Royal Maternity Hospital) of the Royal Infirmary, Edinburgh. The twenty year period, 1928 to 1947 (inclusive) has been reviewed and a follow-up examination of all patients in the ten year period 1937 to 1946 (inclusive) has been carried out. The thesis has been divided into three parts. Part I deals with the review of the past twenty years, Part II with the follow-up examination of patients and Part III with a discussion of the findings in Parts I and II.

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## PART I.

## A REVIEW OF THE PAST TWENTY YEARS.

In 1928 a Cardiac Clinic was started in the Royal Maternity Hospital to deal with the large number of cardiac cases associated with pregnancy and from the records of these cases it has been possible to obtain much valuable information with special reference to the changes both in management and mortality rates that have occurred during this period.

### 1. INCIDENCE OF HEART DISEASE.

Table I shows the total number of cases dealt with in the maternity Hospital, the total number of cardiac cases, the percentage incidence of cardiac cases and the mortality rates. It will be seen that the incidence varies somewhat each year between 0.91 per cent and 1.82 per cent with an average over the twenty years of 1.3 per cent. It is probable that this figure represents a higher percentage than actually occurs among women of the childbearing age mainly because of relatively more cardiac than normal patients being referred to the hospital.

Table II shows the incidence of cardiac cases in Glasgow, Liverpool and Manchester compared with Edinburgh. It is interesting to note that in Glasgow the incidence is much higher either due to more cardiac cases being referred to hospital or

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## TABLE I.

Yearly Figures of Cardiac Cases, Incidence of Cardiac Disease and Mortality Rates, since start of Cardiac Clinic 20 Years ago.

| Year. | Cardiac Pregnancies. | | Deaths. | Mortality Rate per cent. | | |
|---|---|---|---|---|---|---|
| | No. | Per cent Incidence of total Pregnancies. | | All Cases. | Booked Cases. | Non-booked Cases. |
| 1928 | 56 | 1.32 | 2 | 3.5 | 4.6 | 0.0 |
| 1929 | 38 | 0.91 | 3 | 7.7 | 6.9 | 10.0 |
| 1930 | 43 | 1.29 | 1 | 2.3 | 0.0 | 6.6 |
| 1931 | 36 | 1.1 | 3 | 8.3 | 7.0 | 12.5 |
| 1932 | 32 | 0.97 | 3 | 9.4 | 6.2 | 12.5 |
| 1933 | 40 | 1.23 | 6 | 14.6 | 7.5 | 28.5 |
| 1934 | 37 | 1.09 | 3 | 8.1 | 4.3 | 14.2 |
| 1935 | 35 | 1.11 | 2 | 5.7 | 0.0 | 25.0 |
| 1936 | 60 | 1.82 | 3 | 5.0 | 0.0 | 11.0 |
| 1937 | 56 | 1.73 | 3 | 5.1 | 0.0 | 11.0 |
| 1938 | 49 | 1.5 | 2 | 4.1 | 2.9 | 6.7 |
| 1939 | 54 | 1.46 | 1 | 1.9 | 0.0 | 5.9 |
| 1940 | 50 | 1.1 | 1 | 2.0 | 0.0 | 11.1 |
| 1941 | 57 | 1.33 | 3 | 5.1 | 1.9 | 20.0 |
| 1942 | 56 | 1.26 | 2 | 3.6 | 2.6 | 5.0 |
| 1943 | 79 | 1.76 | 0 | 0.0 | 0.0 | 0.0 |
| 1944 | 67 | 1.45 | 3 | 4.2 | 5.1 | 0.0 |
| 1945 | 58 | 1.32 | 1 | 1.7 | 0.0 | 6.2 |
| 1946 | 91 | 1.55 | 3 | 3.3 | 2.8 | 4.5 |
| 1947 | 106 | 1.8 | 1 | 0.9 | 1.2 | 0.0 |
| TOTALS and AVERAGES. | 1100 | 1.3 | 46 | 4.1 | 2.4 | 5.1 |

more likely to an actual increased incidence of rheumatic heart disease in that city.

Jensen (1938) summarising all the available data up to 1937 concludes that the incidence of heart disease amongst obstetric patients is about 1 per cent. This figure varies in different parts of the world and according to the accuracy in diagnosis of heart disease. Jensen states that estimates based on physical examination during pregnancy alone generally appear too high. It has been found that many cases regarded as having heart disease during pregnancy have in fact no demonstrable heart lesion when examined after pregnancy (Part II) so that the actual incidence in Edinburgh is less than 1.3 per cent.

To summarise, roughly one in a hundred women of the childbearing age has heart disease. Figures from different centres vary as a result of three factors. Firstly, cardiac cases may be referred to a hospital thus tending to increase the incidence of these cases. Secondly, methods of diagnosis may vary in different centres. Both these causes tend to increase the incidence. Thirdly, there is probably a geographical variation in the incidence of heart disease encountered during the childbearing years. As 95 per cent of all cardiac cases are due to rheumatic damage it is quite likely that there is

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in fact a higher incidence of this form of heart disease in a damp overcrowded city such as Glasgow, compared with Edinburgh.

TABLE II.

Incidence and Mortality Rates of Cardiac Cases in different Centres over a Period of Twenty Years divided into Two 10-Year Periods.

| Centre. | Cardiac Pregnancies. | | Mortality Rate per cent. | | | |
|---|---|---|---|---|---|---|
| | Total No. | Per Cent Incidence All Pregnancies. | Booked Cases. | | Non-booked Cases. | |
| | | | 1st 10Yrs. | 2nd 10Yrs. | 1st 10Yrs. | 2nd 10Yrs. |
| Edinburgh. | 1100 | 1.3 | 3.5 | 1.7 | 12.6 | 4.4 |
| Glasgow. | 2183 | 2.4 | 4.3 | 2.8 | 9.8 | 7.5 |
| Liverpool. | 730 | 1.7 | 3.7 | 2.2 | 5.4 | 6.2 |
| Manchester. | 1401 | 2.9 | 4.5 | 1.4 | 7.7 | 5.4 |

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2. CARDIAC MORTALITY RATES.

Care must be taken in interpreting maternal mortality figures in cardiac women as the death rates are heavily weighed by women who have

/received.....

received no Ante-natal care and who are admitted in congestive failure. In most hospitals cases are divided into "booked" and "non-booked" (or emergency) cases. It follows that "non-booked" cardiac cases are admitted often in severe congestive failure, fibrillating and generally in bad condition.

It is necessary therefore to separate the mortality rates in these two groups. The relative number of "booked" and "non-booked" cardiac cases varies greatly in different hospitals. In Edinburgh roughly 25 per cent of cases are non-booked whereas in Glasgow and Manchester roughly 50 per cent fall into this category.

Table I shows the number of cardiac deaths each year and the percentage mortality in booked and non-booked cases. In order to appreciate better the trend in the death rate over the 20 years, Table III has been compiled. There the figures are grouped into five year periods. From this table it can be seen that there has been a marked reduction in the number of deaths due to cardiac disease, from about 7 per cent over ten years ago down to about 2 per cent in the past few years. Indeed in 1947, out of 106 cardiac cases only one death was recorded giving a rate of 0.9 per cent. It can also be seen that the incidence of cardiac disease has increased, but this is probably due to increased interest in the

/cardiac.....

cardiac clinic and the referring of a larger number of cardiac cases for opinion, rather than to an actual increase in heart disease.

TABLE III.

Relation of Cardiac Deaths to Maternal Mortality over the past Twenty years at the Simpson Pavilion, Edinburgh.

| Years. | Total Admissions. | Total Deaths. | Maternal Mortality per cent. | Total Cardiac Cases. | Percentage Incidence of Organic Heart Disease. | Total Cardiac Deaths. | Cardiac Deaths as Percentage of Cardiac Cases. | Cardiac Deaths as Percentage of Total Maternal Deaths. |
|---|---|---|---|---|---|---|---|---|
| 1928-32 | 18,359 | 203 | 1.11 | 205 | 1.12 | 13 | 6.3 | 6.4 |
| 1933-37 | 16,499 | 159 | 0.96 | 228 | 1.40 | 17 | 7.5 | 10.7 |
| 1938-42 | 20,015 | 125 | 0.62 | 266 | 1.33 | 9 | 3.4 | 7.2 |
| 1943-47 | 25,549 | 69 | 0.27 | 401 | 1.58 | 7 | 1.7 | 10.2 |
| 1947. | 5,944 | 11 | 0.18 | 106 | 1.80 | 1 | 0.9 | 9.1 |

The figures presented also show a marked decrease in the maternal death rate from all causes, from 1.1 per cent twenty years ago down to 0.18 per cent in 1947. This is almost a tenfold reduction in the rate and is a remarkable achievement. This has been brought about by great advances in the treatment of shock, haemorrhage and pyogenic

/infections....

infections. The greatest drop occurred between the five year periods 1938-42 and 1943-47 - the time when penicillin, sulphonamides and blood transfusion had all become readily available. Naturally the cardiac cases benefitted to a similar extent from these therapeutic measures. On examining the figures however it is apparent that cardiac death rates have not been reduced to such a great extent as the overall rates. In fact there has been roughly a sevenfold reduction as compared with a tenfold.

If we put the cardiac deaths as a percentage of the total deaths this point is brought out more clearly. It will be seen that there has been a relative increase in the number of cardiac deaths from about 6 per cent up to 10 per cent of the total death rate, indicating that cardiac disease is tending to form a larger proportion of the causes of maternal mortality.

Table II shows the cardiac death rates in booked and non-booked cases in Edinburgh, Glasgow, Liverpool and Manchester divided into two ten year periods. It can be seen that a similar reduction has occurred in all the centres and that the death rate in non-booked cases is roughly three times that of booked cases, indicating that ante-natal supervision of cardiac cases is an essential factor in the reduction of the death rate.

Table IV shows the reported death rates in pregnant

/women.....

TABLE IV.

The Reported Death Rates in Pregnant Cardiac Cases from all Sources to date.

| Author. | Year. | No. of Cases. | No. of Deaths. | Per Cent Deaths. |
|---|---|---|---|---|
| | 1890- | | | |
| Jensen. | 1936 | 8995 | 396 | 4.3 |
| Hay. | 1936 | 66 | 1 | 1.5 |
| Henderson. | 1936 | 76 | 2 | 2.6 |
| Hegedorn. | 1937 | 50 | 5 | 10.0 |
| Harris. | 1937 | 100 | 8 | 8.0 |
| Lamb. | 1937 | 102 | 7 | 6.9 |
| McClure. | 1937 | 69 | 3 | 4.3 |
| Naish. | 1937 | 427 | 11 | 2.6 |
| Pardee. | 1937 | 50 | 1 | 2.0 |
| Carr. | 1938 | 44 | 1 | 2.3 |
| Turino and Antony. | 1938 | 102 | 6 | 5.9 |
| Lange. | 1939 | 322 | 6 | 1.9 |
| Clahr Klein and Greenstein. | 1940 | 181 | 4 | 2.2 |
| Jensen Wegner Keys and Smith. | 1940 | 108 | 8 | 7.4 |
| Gorenberg and McGleary. | 1941 | 345 | 10 | 2.9 |
| Hamilton and Thomson. | 1941 | 781 | 37 | 4.7 |
| Bramwell and Longson. | 1942 | 312 | 22 | 7.1 |
| Brown and Sage. | 1942 | 32 | 1 | 3.1 |
| Gorenberg. | 1943 | 223 | 8 | 3.6 |
| Jones. | 1943 | 74 | 4 | 5.4 |
| Sampson. | 1943 | 60 | 0 | 0.0 |
| Mendelson. | 1944 | 1089 | 8 | 0.7 |
| Scott. | 1944 | 114 | 3 | 2.6 |
| Bunim and Rubricius. | 1947 | 142 | 2 | 1.4 |
| Macrae. | 1948 | 335 | 7 | 3.1 |
| Present Series. | 1949 | 1100 | 46 | 4.1 |
| TOTAL and AVERAGES ... | | 15,189 | 607 | 3.9 |

women from all the available sources to date. Jensen, who analysed all the data prior to 1937 gives an average death rate of 4.3 per cent. The average figure including that of Jensen is 3.9 per cent so that the Edinburgh figures compare favourably with any others. Many of these reported figures deal with too few cases to be of significance. For instance Sampson's (1943) series of 60 cases with

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no deaths is very creditable but we could also produce a series of 60 consecutive cases without a death. Mendelson's (1944) series is however noteworthy and the reasons for his low mortality will be discussed later.

It is apparent therefore, taking all the figures into consideration, that even at the present time one or two women out of a hundred with heart disease have succumbed during pregnancy, labour or the puerperum. This is a marked improvement on the figures of twenty years ago, indicating that the methods of management and care of cardiac women during pregnancy and labour have proved most effective. There is, however, no reason why this figure should not be even further improved upon and an essential factor for improvement is adequate ante-natal care.

How improvement has been achieved and how further improvement may be achieved, will be discussed in Part III.

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## 3. THE CAUSES OF DEATH DURING PREGNANCY IN CARDIAC WOMEN.

During the twenty year period under review 46 deaths have occurred out of a total of 1,100 cardiac pregnancies. The case notes of all these fatal cases have been investigated and the cause of

/death.....

death ascertained. There follows a short summary of the case notes of these 46 fatal cases. They have been listed into the main causes of death (i) Con: :gestive Heart Failure, (ii) Acute Pulmonary Oedema, (iii) Bacterial Endocarditis and (iv), (v), (vi) and (vii) Other Causes. The deaths in Congestive Heart Failure have been further subdivided into those occurring (a) in the first half of pregnancy, (b) in the second half of pregnancy (c) following Caesarean Section and (d) following pelvic delivery.

The Grades given are as defined on page 106 Part III.

(i). CASES DYING IN CONGESTIVE FAILURE.
(a). In First Half of Pregnancy. (6 Cases.)

(1) 659/30. Para 3. Age 31. Mitral Stenosis. Several previous attacks of cardiac failure in preceding years. Refused therapeutic abortion. Grade III at 14 weeks. Normal rhythm. Died soon after admission. Autopsy: Advanced Mitral Stenosis with recent simple endocarditis. Pulmonary infarction.

(2) 1163/32. Para 1. Age 38. Mitral Stenosis and aortic incompetence. Normal rhythm. Grade IIb at first ante-natal examination and admitted at once. Died soon after. Autopsy: None.

(3) 1691/36. Para 3. Age 36. Mitral Stenosis. Admitted at 20th week. Grade III with normal rhythm. Digitalised without improvement.

/Auricular.....

Auricular fibrillation and pulmonary oedema developed 10 days later and patient died. Autopsy: None.

(4) 3193/44. Para 1. Age 39. Mitral stenosis. Normal rhythm. Grade III at 15th week. Slight improvement following treatment and termination decided on. Abdominal Hysterotomy with cyclopropane. Patient died two hours after operation. Autopsy: None.

(5) 3148/46. Para 1. Age 25. Mitral Stenosis. Normal rhythm. Grade IIa in 1942 pregnancy. Grade IIa at 6th week of this pregnancy. Grade III at 14th week of this pregnancy. Treatment with digoxin etc., unavailing and patient died two hours after admission. Autopsy: Mitral Stenosis. Cardiac failure with Pulmonary Oedema.

(6) 2237/47. Para 0. Age 37. Mitral Stenosis. Regular ante-natal supervision. Became Grade IIb at 14 weeks gestation. Admitted Grade III and treated for five weeks with rest, digitalis and neptal. Condition then deemed optimum for interruption of pregnancy. Supra-vaginal Hysterectomy. Anaesthetic spinal, pentothal, cyclopropane and oxygen. Sudden death 16 hours after operation. Autopsy: None.

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(b). In Second Half of Pregnancy. (6 Cases.)

(1) 865/29. 2 Miscarriages. Age 27. Mitral Stenosis. Normal rhythm. Previous attacks of cardiac failure. Grade III at 32nd week. No response to treatment and death 27 hours after admission. Autopsy: None.

(2) 498/33. Para 2. Age 45. Mitral Stenosis and Aortic Incompetence. Grade IIb at 22nd week. Normal rhythm. 5 weeks later developed bronchitis and gradually deteriorated. Death at 30th week. Autopsy: None.

(3) 1010/34. Para 0. Age 26. Mitral Stenosis. Grade IIb from 2nd to 5th month (in bed). Grade III at 26th week. Responded to rest and digitalis. Abdominal Hysterotomy performed under local anaesthesia. 4 days later patient collapsed with severe precordial pain, cyanosis and dyspnoea and died 6 hours later. Autopsy: None.

(4) Para 0. Age 24. Mitral Disease and Chorea. Previous attacks of Chorea. Grade III at 28th week with normal rhythm. Severe Chorea gravidarum. No response to intravenous digoxin and patient died two hours after admission undelivered. Autopsy: Confluent Broncho: :pneumonia. Old and acute rheumatic endocarditis.

(5) 269/38. Para 2. Age 25. Mitral Disease. Grade IIb since 3rd month. Normal rhythm.

/Admitted.....

Admitted at 27th week. Grade III. No improvement to treatment and died 6 days after admission. Autopsy: Grossly fibrosed mitral valve (diameter ¼ inch) with in addition recent vegetations.

(6) 41/44. Para 2. Age 33. Mitral Stenosis. Grade III at 25th week. Slight improvement under treatment not maintained. Miscarried at 28th week followed by severe congestive failure and died 14 hours after Miscarriage. Autopsy: None.

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(c). Following Caesarean Section. (8 Cases.)

(1) 783/31. Para 1. Age 32. Mitral Stenosis. Grade III at 30th week. Normal rhythm. Good response to treatment but left hospital against advice. Readmitted at 34th week Grade III. Some response to treatment but not complete. Caesarean Section at 38th week. Acute cardiac distress and death 29 hours after delivery. Autopsy: None.

(2) 1596/32. Para 0. Age 33. Auricular fibrillation and probably Mitral Stenosis. Grade III at 29th week. Improved for 3 weeks then relapsed in 4th week to Grade III. Caesarean Section under local anaesthesia at 33rd week. Developed bronchitis and died 24 hours after operation. Autopsy: None.

/(3).....

(3) 1652/32. Para 10. Age 40. Mitral Stenosis. Admitted Grade III at 32nd week. 3 days later Caesarean Section under local anaesthesia. Developed Broncho-pneumonia 2 days later and after 8 days wound gave way - resutured under chloroform. Died 24 hours later.
Autopsy: None.

(4) 67/33. Para 10. Age 42. Mitral Stenosis. Admitted Grade III at 27th week. Digitalised and improved. Caesarean Section 1 month later under local anaethesia - good recovery. 18 days later sudden cyanosis, dyspnoea and death.
Autopsy: Ball Thrombus in (L) auricle blocking stenosed mitral orifice.

(5) 318/34. Para 1. Age 23. Mitral Stenosis. Normal rhythm. History of haemoptysis during pregnancy. Admitted Grade III at 40th week and 2 hours later Caesarean Section performed under local anaesthesia supplemented by chloroform and oxygen. Collapsed and died 2 hours after operation. Autopsy: None.

(6) 964/41. Para 2. Age 33. Mitral Stenosis. Normal rhythm. Admitted Grade III at 35th week. Little improvement from treatment and Caesarean Section performed 3 weeks later. Died in a few hours. Autopsy: None.

(7) 2652/41. Para 0. Age 32. Mitral Stenosis. Admitted Grade III at 32nd week. Caesarean

/Section.....

Section 2 weeks later under local anaesthesia. Condition deteriorated and wound burst on 7th day and she died 7 hours after resuturing. Autopsy: None.

(8) 2890/44. Para 1. Age 27. Mitral Stenosis. Normal rhythm. Grade III at 24th week but improved to IIa. Readmitted Grade III at 30th week. Initial response to treatment good but 6 weeks later sudden deterioration with appearance of congestive heart failure. Caesarean Section decided on to alleviate condition. Cyclopropane anaesthesia. Death 12 hours after operation. Autopsy: None.

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(d). Following Pelvic Delivery. (8 Cases).

(1) 1010/33. Para 1. Age 25. Mitral Stenosis. Normal rhythm. Haemoptysis 2 weeks before admission. Admitted Grade III at 28th week. Venesection. Spontaneous delivery next day. Became collapsed and no response to oxygen and digitalis. Died 15 hours after delivery. Autopsy: None.

(2) 2034/34. Para 6. Age 40. Mitral Stenosis and auricular fibrillation. Previous failure before pregnancy. Admitted Grade III at 32nd week with auricular fibrillation. Medical induction at 36th week after improvement. Sudden death 4 days after labour. Autopsy: None.

/(3).....

(3) 1311/35. Para 1. Age 34. Mitral Disease. Normal rhythm. Admitted Grade III at 34 weeks. Digitalised. Next day spontaneous 1½ hour labour with deterioration of heart condition. Died 6 hours after labour. Autopsy: Acute and chronic rheumatic endocarditis of mitral valve. Mitral Disease.

(4) 1468/35. Para 0. Age 27. Mitral Disease. Admitted Grade III at 35th week. Previous failure and haemoptysis. Digitalised. Spontaneous labour 3 weeks later lasting 15 hours. Auricular fibrillation developed after labour and death took place 7 days later - congestive heart failure. Autopsy: Chronic and acute endocarditis with infarcts in lungs.

(5) 57 /36. Para 1. Age 36. Mitral Stenosis. Normal rhythm. Grade III at 6 months. Readmitted serious Grade III at 36th week, in labour. Digitalised and transfused with blood. Delivered herself in 1½ hours but collapsed 1½ hours later. Autopsy: Marked Chronic Mitral Stenosis with recent warty vegetations. Hydrothorax.

(6) 2807/39. Para 0. Age 40. Mitral Stenosis and auricular fibrillation. Admitted Grade III at 23rd week fibrillating. Digitalised and improved. 12 weeks later, after marked improvement, spontaneous rupture of membranes,

/and.....

and low forceps delivery after 2 hour labour. Died almost immediately after labour with cardiac and respiratory failure. Autopsy: None.

(7) 1452/41. Para 0. Age 25. Mitral Stenosis. Normal rhythm. Grade III at mid-pregnancy and in addition active Pulmonary Tuberculosis and Pyelitis. Readmitted at 28th week Grade III. Labour began spontaneously at 38th week and lasted 46 hours. She stood delivery well but on 6th day she had sudden pain in chest, dyspnoea and haemoptysis and died 24 hours later. Congestive failure and pulmonary embolism. Autopsy: None.

(8) 159/42. Para 1. Age 39. Mitral Disease. Normal rhythm. Admitted Grade III at 29th week with negligible response to treatment. Spontaneous labour lasting 1½ hours, 10 days later. Died immediately after delivery. Autopsy: None.

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(ii). CASES DYING AS A RESULT OF ACUTE PULMONARY OEDEMA.

By this is meant cases dying suddenly without previous evidence of congestive heart failure and in whom there was acute left sided failure causing pulmonary oedema. (6 Cases).

(1) 1108/31. Para 0. Age 28. Mitral Stenosis. Normal rhythm. Grade I throughout pregnancy.

/Admitted.....

Admitted Grade I at 38th week. Labour commenced 4 days later. Sudden distress during second stage with no response to treatment. Died during second stage. Autopsy: None.

(2) 208/33. Para 1. Age 43. Mitral Stenosis. Auricular fibrillation. Treated at 5th month for auricular fibrillation, broncho-pneumonia and hyperemesis gravidarum. No congestive failure. Readmitted at 31st week with attacks of dyspnoea but Grade IIa. Spontaneous labour one week later lasting 3 hours. Three hours later developed acute pulmonary oedema and died shortly after. Autopsy: Lungs congested and oedematous. Marked Mitral Stenosis.

(3) 1095/33. Para 2. Age 30. Mitral Stenosis. Normal rhythm. History during pregnancy of sickness, cough, haemoptysis and headaches. Admitted at 30 weeks with hyperemesis gravidarum. Digitalised. Sickness stopped. Spontaneous onset of labour which lasted twenty minutes and ended naturally two days later. Sudden collapse and death 2 hours following delivery and despite treatment. Autopsy: None.

(4) 1603/40. Para 0. Age 19. Mitral Stenosis and aortic incompetence. Normal rhythm. Grade IIa throughout pregnancy. 10 days before term developed acute dyspnoea and haemoptysis. No

/response....

response to digoxin and mercurial diuretics and died 18 hours after admission. Autopsy: Pulmonary oedema with recent vegetations on old mitral and aortic rheumatic endocarditis.

(5) 290/45. Para 3. Age 37. Mitral Stenosis. Normal rhythm. Treated for general debility with iron tonics. Spontaneous labour at term lasting 4½ hours with slight haemorrhage. Collapsed one hour after delivery but rallied. Collapsed 24 hours later with dyspnoea and cyanosis and died. Autopsy: None.

(6) 3148/46. Para 1. Age 25. Mitral Stenosis. Normal rhythm. Grade IIa in 1942. Grade IIa in this pregnancy. Admitted at 14th week with acute pulmonary oedema, with no response to treatment and died 2 hours after admission. Autopsy: Old and recent rheumatic carditis. Mitral Stenosis. Pulmonary oedema.

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(iii). CASES DYING AS A RESULT OF BACTERIAL ENDOCARDITIS. (7 Cases).

(1) 1693/28. Para 1. Age 25. Mitral Disease. Normal rhythm. No history of previous rheumatic heart lesion. Medically induced labour because of albuminuria. Spontaneous delivery. Puerperal pyrexia developed on second day and patient died 16 days later from "ulcerative" endocarditis. Autopsy: None.

/(2).....

(2) 1625/31. Para 5. Age 37. Mitral Stenosis and incompetence. Normal rhythm. Rheumatic fever 4 years previously. Grade IIb at 15th week. Admitted Grade III at 20th week with marked anaemia. Unsatisfactory response to digitalis and liver therapy. Blood transfused, and pregnancy terminated by subtotal hysterectomy. Pulmonary oedema developed and death occurred 24 hours after operation. Autopsy: Old standing Mitral Stenosis and incompetence with superimposed vegetations of recent acute malignant endocarditis.

(3) 1057/33. Para 0. Age 26. Mitral Stenosis. Normal rhythm. Treated twice for marked dyspnoea and tachycardia. Admitted at 30th week Grade IIa. 2 weeks later Caesarean Section performed under local anaesthesia. 3 days after, high temperature developed and death occurred 6 days after operation. Autopsy: Mitral Stenosis with eroded valves (ulcerative endocarditis). Pyaemic infarcts in various organs. Causative organism, Staphylococcus aureus.

(4) 91/37. Para 0. Age 21. Mitral Stenosis. Normal rhythm. Developed a cold 14 days before admission and became progressively more dyspnoeic. Admitted at 39th week. Normal

/temperature.....

temperature. Mitral Stenosis and pre-eclamptic toxaemia. Caesarean Section under local anaesthesia performed on second day. From then on temperature and pulse rose and signs of endocarditis became apparent. Death on 19th day. Autopsy: Acute ulcerative endocarditis and infarct in left lung.

(5) 2353/37. Para 2. Age 34. Mitral Stenosis. Normal rhythm. Developed a "cold" at 31st week of gestation and became very dyspnoeic. Admitted at 33rd week with normal temperature, marked Mitral Stenosis and bronchitis with crepitations at both bases. No improvement during next week and patient collapsed and died on the 8th day after admission. Autopsy: Mitral Stenosis with superadded subacute bacterial endocarditis. Bilateral Lobar Pneumonia. Two old infarcts in spleen.

(6) 2047/38. Para 2. Age 28. Mitral Stenosis. Normal rhythm. Had been in bed at home with raised pulse and temperature and some cough. Given sulphonamides 5 days before admission. Admitted at 20th week, pulse 140, temperature 99.8, and Mitral Stenosis, Grade III. Gradually deteriorated over the next 17 days with a temperature first of a high and then of a low remittent type. Jaundice developed and dyspnoea and general distress became worse.

/Died.....

Died 17 days after admission. Autopsy: Mitral and aortic valves showed old endocarditis with superimposed very gross subacute bacterial endocarditis with large vegetations. Early focal embolic nephritis.

(7) 2126/42. Para 0. Age 36. Mitral disease. Mild pre-eclamptic toxaemia before going into spontaneous labour. Inertia led to labour of 85 hours, to midcavity forceps delivery and manual removal of placenta. Puerperal pyrexia developed but subsided with chemotherapy and blood transfusion but she developed septic lung infarcts and acute bacterial endocarditis which proved fatal a month later. Autopsy: Confirmed acute bacterial endocarditis.

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(iv). DEATH DUE TO ACUTE RHEUMATIC FEVER.

1392/29. Para 0. Age 22. Chorea and acute endocarditis. Admitted at 36th week with chorea and commencing cardiac failure but deteriorated with sudden onset of dyspnoea, coughing and epigastric pain 18 days later and died. Autopsy: Acute mitral endocarditis. Acute myocarditis. Congestive failure.

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(v). DEATH DUE TO ANAESTHESIA.

774/29. Para 2. Age 35. Mitral Stenosis.

/Normal.....

Normal rhythm. Grade IIb at 32nd week successfully treated. Admitted at 31st week in labour - precipitous. Died of syncope during induction of chloroform anaesthesia to repair a perineal tear. Autopsy: None.

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(vi). DEATHS DUE TO PERITONITIS.

(1) 1456/28. Para 1. Age 23. Mitral Stenosis. Normal rhythm. Grade IIa throughout pregnancy but admitted for Caesarean Section and sterilisation. Died 4 days after operation - acute peritonitis. Autopsy: None.

(2) 918/37. Para 2. Age 30. Chorea, endocarditis and peritonitis. Slight choreiform movements for five months. Severe for 2 weeks prior to admission. Admitted at 28th week with severe chorea. No improvement following treatment and Caesarean Section performed. 5 days later developed peritonitis and died next day. Autopsy: Acute rheumatic endocarditis. Acute peritonitis.

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(vii). DEATH DUE TO HAEMOGLOBINURIA NEPHROPATHY.

3102/46. Para 0. Age 35. Mitral Stenosis. Normal rhythm. Grade IIa throughout pregnancy. Placenta praevia with severe haemorrhage requiring several blood transfusions. Oliguria

/and.....

and jaundice developed with no response to treatment. Died 9 days after delivery. Autopsy: Mitral Stenosis and Haemoglobinuria nephropathy.

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In Table V the main causes of death are listed.

TABLE V.

Causes of Death in the 46 Fatal Cases.

| Cause of Death. | No. of Cases. | Per Cent of Total. |
|---|---|---|
| Congestive Heart Failure. | 28 | 61 |
| Bacterial Endocarditis. | 7 | 16 |
| Acute Pulmonary Oedema. | 6 | 13 |
| Peritonitis seq. Caesarean Section. | 2 | 4 |
| Acute Rheumatic Fever. | 1 | 2 |
| Anaesthetic. | 1 | 2 |
| Haemoglobinuria Nephropathy. | 1 | 2 |
| TOTAL ... | 46 | 100 |

It can be seen that the great majority die as a result of congestive heart failure (61 per cent) and that the next most important cause is bacterial endocarditis which accounts for 16 per cent of all fatal cases. There were seven cases in this group and five of these developed acute ulcerative endocarditis in the course of puerperal fever. The

/other.....

other two cases had subacute bacterial endocarditis which developed probably during the pregnancy. The next most important cause of death is acute pulmonary oedema or acute heart failure occurring in the absence of "chronic" congestive heart failure. This group accounts for 13 per cent of all the cases. Two cases died as a result of generalised peritonitis following Caesarean Section, one died as a result of acute rheumatic fever with carditis, one died as the result of administering chloroform anaesthesia and one died as a result of incompatible blood transfusion, necessitated by severe post partum haemorrhage.

It is interesting to note that no case has pulmonary embolism recorded as the cause of death. One or two of the cases dying in congestive failure probably had a terminal pulmonary infarct e.g., Case No. 1010/34 but these cases have been listed under congestive heart failure.

Table VI shows the causes of death in Hamilton's series of 54 fatal cases (Hamilton and Thomson, 1941). It will be noted that there is a high incidence of pulmonary embolism as a cause of death - 14.8 per cent of cardiac origin. It may be that these cases had congestive heart failure and the pulmonary infarct was the terminal event. He also shows a lower incidence of bacterial endocarditis and no cases had acute pulmonary oedema, though the two

/cases.....

cases dying suddenly may probably have died from this latter cause.

TABLE VI.

Causes of Death in Hamilton's Series of 54 Cases.

| Cause of Death. | No. of Cases. | Per Cent of Total. |
|---|---|---|
| Congestive Heart Failure. | 31 | 57.4 |
| Embolism (Cardiac origin). | 8 | 14.8 |
| Pulmonary embolism (peripheral origin). | 3 | 5.5 |
| Subacute Bacterial Endocarditis. | 3 | 5.5 |
| Acute Rheumatic Fever. | 2 | 3.7 |
| Influenza. | 2 | 3.7 |
| Sudden. | 2 | 3.7 |
| Combined Disease. | 1 | 2.0 |
| Unknown Cause. | 2 | 3.7 |
| TOTAL .... | 54 | 100 |

Of the 46 deaths twenty had a post mortem examination. In Table VII the findings in the heart valves are recorded.

TABLE VII.

Autopsy Findings on 20 Cases.

| Post-mortem Findings. | No. of Cases. | Per cent of Total. |
|---|---|---|
| Recent acute rheumatic vegetations on chronic. | 11 | 55 |
| Acute and subacute bacterial endocarditis on chronic. | 6 | 30 |
| Chronic rheumatic endocarditis only. | 3 | 15 |
| TOTAL .... | 20 | 100 |

/It.....

It will be seen that over half the cases showed recent acute rheumatic vegetations superimposed on chronic damage and that in only 15 per cent of the cases was no evidence of recent activation found. 30 per cent showed evidence of bacterial endocarditis (acute and subacute) on chronic valvular damage. Although no great significance can be attached to these findings as less than half the cases had autopsy examination it is noteworthy that over half the cases on which post-mortem examination was made showed evidence of recent rheumatic activation when no clinical evidence was apparent. This finding brings out the point that rheumatic cardiac infection can be active without clinical evidence of rheumatic fever, or evidence of the rheumatic state. In only one of these cases was acute rheumatic fever present clinically.

It is interesting and instructive to list the maternal deaths in relation to the duration of pregnancy and to delivery. In Table VIII the main causes of death have been separated into their relationship to the time of pregnancy - before mid-term, after mid-term, during labour and in the puerperium. It will be seen that 60.8 per cent of the fatal cases occur in the puerperium and that only 2.2 per cent die during labour. The deaths during pregnancy are about equally distributed before and after mid-term.

/Table VIII.....

TABLE VIII.

The Distribution of the 46 Deaths.

| Cause of Death. | Before Mid-term. | After Mid-term. | Labour. | Puer: perium. |
|---|---|---|---|---|
| Congestive Heart Failure. | 6 | 6 | 0 | 16 |
| Acute Pulmonary Oedema. | 1 | 1 | 1 | 3 |
| Bacterial Endocarditis. | 0 | 2 | 0 | 5 |
| Other Causes. | 0 | 1 | 0 | 4 |
| TOTAL ..... | 7 | 10 | 1 | 28 |
| PERCENTAGE ..... | 15.2 | 21.8 | 2.2 | 60.8 |

If we analyse these figures more fully (as is done in the list of fatal cases) we find that there were no deaths from congestive heart failure during the first three months of pregnancy with the possible exception of Case No. 1163/32 and none in the last month. Nearly all the deaths from this cause occurred between the fifteenth and thirtieth weeks. It is well known that the burden of pregnancy becomes marked from the third month onwards and that there is a reduction of cardiac work during the last month of pregnancy. It is unlikely that fatal congestive failure will be apparent before the third month and if the heart has managed to respond to the increasing demands made upon it up till the ninth month, it is unlikely that it will fail in the final month. These figures would appear to support these contentions.

No case died in congestive heart failure

/during.....

during labour. The puerperium however, accounted for two-thirds of the deaths in congestive heart failure - 16 cases in all. 8 of these cases died following normal or assisted delivery and 8 died following Caesarean Section. Of the 16 cases 10 died within 24 hours, (actually one of those died at 27 hours), 6 of them within a few hours of delivery. Of the 6 remaining, 2 cases, delivered by Caesarean Section, burst their wounds a few days after operation and died several hours later, 3 cases had sudden pulmonary infarcts at the 4th, 6th and 7th days respectively and 1 case died (18 days after delivery) as a result of a ball thrombus blocking the stenosed mitral orifice. The average age at death from this cause was 34 years. 10 died in their first pregnancy and 18 had previous pregnancies. The average age of the primipara was 31 years and of the multipara 34.5 years. 6 cases showed auricular fibrillation and their average age was 36 years.

Of the 6 cases dying in acute pulmonary oedema, 1 occurred at the fourteenth week, 1 occurred 10 days before term, 1 occurred during the second stage of labour and 3 occurred within a few hours of delivery. The case numbered 208/33 was noted to have auricular fibrillation at the fifth month and it could be argued that the sudden death was due to a pulmonary embolism. However none was found on post-mortem

/examination.....

examination, the main finding being pulmonary oedema. The case is therefore classified under the heading of acute pulmonary oedema.

None of these cases had at any time exhibited signs of congestive heart failure and their deaths were all very sudden, and without warning. In 2 cases however a previous haemoptysis had occurred and in one there was a history of attacks of dyspnoea a few weeks prior to death. 2 of the cases were complicated by hyperemesis gravidarum. The average age of this group (excluding the case of auricular fibrillation) was 25.5 years i.e., 9 years less than the age at death in congestive failure.

Of the 7 cases dying as a result of bacterial infection, 4 cases developed the infection during puerperal fever, 2 of them following operative interference, and each showed clinical signs of acute ulcerative endocarditis. 1 case developed signs of ulcerative endocarditis during pregnancy and died after subtotal hysterectomy. 2 cases developed subacute bacterial endocarditis during pregnancy and both died before delivery. The diagnosis was confirmed at autopsy in 6 of the 7 cases.

Of the deaths listed under other causes, all but 1 died in the puerperium; 2 as a result of peritonitis following Caesarean Section, one during anaesthetic induction for a perineal repair, and 1 following incompatible blood transfusion. The

/remaining.....

remaining case died just before term as a result of acute rheumatic fever and chorea with acute endocarditis.

To sum up then, congestive heart failure is the commonest cause of death and death from this cause most frequently occurs very soon after delivery, often within a few hours of the end of labour. During labour itself death is very unlikely to occur. A fatal termination from any cause is much more common in the puerperium - almost two thirds of the deaths occurring during this period.

Excluding cases of bacterial endocarditis, which nowadays are largely preventable, and cases listed under other causes (and taking into account only cases of congestive heart failure and acute pulmonary oedema) the most critical time for a cardiac woman is the few hours immediately following delivery. The reason for this observation is difficult to explain. The labour itself appears to be overcome and yet something happens in the next few hours which causes the already damaged heart to fail completely. It may be that there is a sudden increase in the volume of circulating blood due to the contraction of the uterus expelling all the blood from its sinuses thus causing an "overloading" of the circulation analagous to a transfusion given too rapidly. Sudden death immediately following delivery can occur in women with apparently normal hearts. In Table IX

/are.....

are listed 11 cases which had died and the cause of death had been attributed to cardiac failure.

TABLE IX.

The Causes of Death in 11 Cases which had been Classified under Heart Disease.

| Cause of Death. | No.of Cases. | Remarks. |
|---|---|---|
| Pre-eclamptic Toxaemia. | 4 | All had acute left ventricular failure. |
| Sudden. | 4 | No obvious cause. Post-mortem reports given. |
| Heart Failure. | 2 | Following Pneumonia.<br>" Dehydration. |
| Purulent Pericarditis. | 1 | Spread from Pneumonia. |

It can be seen that 4 cases of pregnancy toxaemia died, all as a result of acute left ventricular failure, 2 died in heart failure secondary to acute lobar pneumonia and dehydration respectively and 1 died with purulent pericarditis secondary to pneumonia.

There remain 4 cases all of whom died suddenly. A summary of these cases is given as they form a very interesting group. All but one had postmortem examinations.

(1) 1232/29. Para 0. Age 22. Acute cardiac failure. Died with acute dyspnoea one hour after normal delivery. Autopsy: Cardiac dilatation but no myocardial degeneration. The aorta was very narrow. Other organs healthy.

/(2).....

(2) 257/37. Para 4. Age 31. Acute cardiac failure. Collapsed during labour, became cyanosed dyspnoeic and died immediately. Autopsy: Generalised cardiac dilatation, pale soft myocardium with small endocardial haemorrhages in left ventricle.

(3) 2340/41. Para 0. Age 28. Acute cardiac failure and pulmonary oedema. Minor degree of cardiac distress antenatally. Delivered at term by low forceps because of great distress with cyanosis. Cyanosis persisted after delivery and pulmonary oedema developed. Died 8 hours after delivery.
Autopsy: None.

(4) 1703/44. Para 0. Age 38. Myocarditis and acute heart failure. Uneventful pregnancy. Maternal distress during labour and low forceps used. Patient collapsed after delivery with acute pulmonary oedema. Autopsy: Cardiac adiposity and acute heart failure.

One of these cases died during labour and the other three died within a few hours of delivery. None had a clinically detectable organic lesion and they each had signs suggesting acute pulmonary oedema. Only one had symptoms referable to the cardiovascular system antenatally but no more than might have been expected in a normal woman. One case may have had congenital hypoplasia of the aorta but the others presented no definite postmortem finding that could

/account.....

account for heart failure. Again it may be that the contractions of the uterus increased suddenly the amount of circulating blood causing acute heart failure and pulmonary oedema.

A further discussion of this point will be given later in Part III.

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4. INCIDENCE OF TERMINATIONS.

Over the past twenty years many changes have been made in the management of pregnant cardiac women. Knowledge has been gained and this knowledge applied to the treatment of cases. Most of these changes cannot be tabulated. A study can be made however on the extent to which termination of pregnancy has been carried out and also on the method of delivery of cases going to term. A study of these items would be of interest in interpreting the marked improvement in cardiac mortality in the past years. In Table X is shown the percentage of terminations in each four year period.

TABLE X.

Percentage of Cardiac Cases in which Pregnancy was terminated over 20 years, divided into 4-year Periods.

| Four-year Period. | 1928-1931. | 1932-1935. | 1936-1939. | 1940-1943. | 1944-1947. |
|---|---|---|---|---|---|
| Percentage Cases terminated. | 6.2 | 14.3 | 6.9 | 11.3 | 10.7 |

/It.....

It can be seen that overall there has been very little change in the past twenty years roughly 10 per cent of women having their pregnancy terminated.

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5. CAESAREAN SECTION: INCIDENCE AND MORTALITY RATES AS COMPARED WITH PELVIC DELIVERY.

Table XI shows the percentage number of cases delivered by Caesarean Section in each four year period in the four centres - Edinburgh, Glasgow, Liverpool and Manchester.

TABLE XI.

Percentage of Cardiac Cases delivered by Caesarean Section in different Centres over the past 20 Years divided in four-year Periods.

| | 1928-31 | 1932-35 | 1936-39 | 1940-43 | 1944-47 |
|---|---|---|---|---|---|
| Edinburgh. | 14.9 | 11.0 | 9.9 | 13.0 | 9.8 |
| Glasgow. | 14.3 | 9.0 | 9.2 | 6.0 | 4.7 |
| Liverpool. | 14.2 | 29.4 | 41.1 | 31.0 | 22.7 |
| Manchester. | 4.6 | 5.3 | 8.5 | 5.0 | 4.4 |

These figures make interesting study. Twenty years ago the number of cases delivered by Caesarean Section in Edinburgh, Glasgow and Liverpool was around 14 per cent while in Manchester it was only 4.6 per cent. It will be seen that Manchester has a persistently low percentage over the twenty years whereas Liverpool has a persistently high percentage.

/Glasgow.....

Glasgow shows a tendency to decrease the number of cases delivered by Caesarean Section and this tendency is apparent in Edinburgh to a less degree. From the wide variation in the figures from different centres it is obvious that the indications for Caesarean Section must vary between these centres.

It had always been thought that a woman with heart disease might not stand up to the increased efforts of labour and accordingly any means of shortening or alleviating this stress would be an advantage. Consequently in severe cardiac cases it was considered that Caesarean Section was indicated. However over a period of years it became apparent that these cases were not doing well following operative interference and many deaths were being recorded. Recently the tendency has been to perform Caesarean Section less and less. This is the current practice but it has not been possible to give figures to prove this point from the information collected from the four centres, with the possible exception of Glasgow which has shown a steady decrease over the past twenty years in the number of Caesarean Sections performed.

If Caesarean Section were not justified it would be expected that the mortality rates following Caesarean Section would be higher than following Pelvic delivery - other factors being the same - and that in the centres where Caesarean Section was

/performed.....

performed in a larger number of cases the mortality rates would consequently be higher.

In Table XII are presented the figures from the four centres over twenty years showing the number of cardiac cases, the number and percentage of them delivered by Caesarean Section and the number of deaths following Caesarean Section.

TABLE XII.

Incidence of Caesarean Section among Cardiac Women and Mortality Rate following Section in different Centres over Twenty Years.

| Centre. | No. of Cardiac Cases. | Caesarean Section. | | Deaths. | |
|---|---|---|---|---|---|
| | | No. | Per Cent. | No. | Per Cent. |
| Edinburgh. | 1100 | 124 | 11.3 | 11 | 8.8 |
| Glasgow. | 2183 | 185 | 8.4 | 18 | 9.7 |
| Liverpool. | 730 | 197 | 27.0 | 7 | 3.5 |
| Manchester. | 1401 | 76 | 5.5 | 6 | 7.8 |

It is extraordinary that in Liverpool where over one quarter of cases have had Caesarean Section the lowest mortality rate is shown and that Manchester with the lowest number of Caesarean Sections has a mortality rate only slightly below Edinburgh and Glasgow but over twice as high as in Liverpool. From these figures it can only be assumed that Manchester has been confining Caesarean Section to bad risk cases

/and.....

and that Liverpool has been less particular in its choice of cases for delivery by this method. In other words these figures are not of much value as we are not comparing similar cases. They do show however that the mortality rate following Caesarean Section is between 3.5 and 9.5 per cent.

In order to compare the mortality rates following Caesarean Section with those following Pelvic delivery, it is necessary to be dealing with exactly similar groups of cases. It is very difficult, if not impossible, to obtain two such groups. Previously Caesarean Section was especially indicated in cases with congestive heart failure, cases that were obviously bad risks whichever method of delivery was carried out, and consequently the mortality rate would be expected to be higher. Less severe cases would probably have been allowed to deliver themselves spontaneously. In spite of this disadvantage in comparison, the Edinburgh series of cases has been analysed and only severe cardiac cases (Grades IIb and III) have been compared with respect to mortality rates following Caesarean and Pelvic delivery. These figures are shown in Table XIII and are compared with results obtained by other authors. Very few such studies have been made and these recorded are the only ones that could be found in the literature. In the Edinburgh series the mortality rate following Caesarean Section is 12.4

/per.....

per cent as compared with 6.8 per cent following Pelvic delivery.

TABLE XIII.

Mortality Rates following Caesarean Section and following Pelvic Delivery in Different Centres.

| Centre. | Caesarean Section. | | | Pelvic Delivery. | | |
|---|---|---|---|---|---|---|
| | | Deaths. | | | Deaths. | |
| | Total. | No. | Per Cent. | Total. | No. | Per Cent. |
| This Series (1949) ) Grades ) IIb & | 81 | 10 | 12.4 | 176 | 12 | 6.8 |
| Mendelson (1944) ) III only. | 21 | 2 | 9.5 | 101 | 0 | 0 |
| Gorenberg and McGleary (1941) | 29 | 4 | 13.8 | 315 | 6 | 1.9 |
| Hamilton and Thomson (1941) | 180 | 15 | 8.3 | 706 | 4 | 2.0 |

Unfortunately, due to insufficient number of cases, these figures cannot be regarded as statistically significant but they do suggest that the mortality rate is twice as high following Caesarean Section. Similar differences are shown in the figures from other Centres. All go to prove the contention that Caesarean Section is not so safe a method of delivery as Pelvic delivery even in severe cardiac disease. Although there is no scientific proof, all the evidence and clinical experience go to support this finding. The reasons why pelvic delivery would appear to be safer, will be discussed later.

## PART II.

## THE FOLLOW-UP EXAMINATION.

### 1. NUMBER OF CASES EXAMINED AND TYPES OF HEART DISEASE ENCOUNTERED.

The 10 year period 1937 to 1946 inclusive was selected and an attempt was made to find out the fate of all the cardiac cases passing through the Simpson Pavilion during this period. In all, there were about 400 new cardiac cases during this period. (This figure does not compare with the sum of the numbers listed in the annual reports where a cardiac case is listed each time pregnancy occurs.) Excluding from this number women who died during pregnancy and several unmarried women on whom a follow-up examination was not attempted, 365 cases were left for the purpose of the examination. All but 13 or 4 per cent of these were traced. It must be remembered that during this period there was large scale movement of population and the 13 untraced cases were women who were in the Edinburgh area for a short time and who have now left the district. There is no reason to suppose that they remained untraced because they had died. 352 cases were traced. The majority of these were examined in the Royal Infirmary where a careful history was taken with special reference to rheumatic infection and functional capacity before, during and after pregnancies. A few

/were.....

were examined in their homes and information regarding these cases living a long distance from Edinburgh was obtained from the patient's own doctor. 41 cases were found to have died and the cause of death in each case was obtained from the Registrar General in Edinburgh.

All these 352 cases were classified as cardiacs during one or other of their pregnancies but on follow--up examination 37 (10.2 per cent) were found to have no cardiovascular abnormality. Table XIV shows these figures.

TABLE XIV.

Incidence and Types of Heart Disease on Follow-up Examination of 352 Women regarded as Cardiac Subjects during Pregnancy.

| Etiology. | Incidence per Cent. |
|---|---|
| Rheumatic Heart Disease. | 85.0 |
| Negative. | 10.2 |
| Congenital. | 2.6 |
| Miscellaneous. | 2.2 |

The remaining 315 cases all had heart disease. They have been divided into rheumatic heart disease, congenital heart disease, hypertensive heart disease and miscellaneous cardiac states. 94 per cent of these cases have rheumatic heart disease. Table XV shows the figures.

/TABLE XV.....

TABLE XV.

Percentage Distribution of the Types of Organic Heart Disease encountered among 315 Cardiac Women.

| Etiology. | Incidence per Cent. |
|---|---|
| Rheumatic Heart Disease. | 94.0 |
| Congenital Heart Disease. | 3.6 |
| Hypertensive Heart Disease. | 1.8 |
| Miscellaneous. | 0.6 |

All the 41 deaths occurred in women with rheumatic heart disease. Since the rheumatic group is by far the largest, detailed analysis will be carried out on this group.

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2. RHEUMATIC HEART DISEASE.

In this series rheumatic heart disease comprises 94 per cent of all organic heart disease in pregnancy. This figure is almost the same as quoted by other workers. All give a figure between 90 and 95 per cent. The series comprises 295 cases all of whom had one, or more, pregnancy during the period 1937-1946 and includes only those women who survived pregnancy and the puerperium and who were discharged from the Royal Simpson Pavilion in a fit state. 254 cases were found to be alive in various states of

/health.....

health varying from fully compensated cases to cardiac cripples and 41 cases were found to have died during the period.

Of those examined all were questioned about history of rheumatic infection, chorea, growing pains, scarlet fever, tonsillitis and sore throats and the age of occurrence of these infections noted. All were questioned about functional ability both during and after pregnancy and the case notes consulted with this in view. In all cases the heart was ausculated to determine the valvular lesion, and evidence of congestive failure sought in the lung bases and the ankles. A blood pressure examination was carried out and the majority of cases were X-rayed in the anterior and right oblique views and electrocardio: :graphic tracings were done. Their functional capacity at the time of examination was also recorded with special reference to their ability to carry out daily duties of housework, shopping etc. The grades are as given on Page 106.

All cases were questioned re cough, haemoptysis and palpitation. Any marked increase in weight was noted.

(a). <u>THE RHEUMATIC INFECTION.</u>

At the outset one is faced with the difficulty of knowing what is the rheumatic state, how does it show itself clinically, what is the

/date.....

date of its onset and how often is the heart permanently affected. Many cases of advanced rheumatic heart disease give no history of an acute episode of rheumatic fever, and, conversely, many cases of acute rheumatic fever appear to suffer no permanent cardiac disability. We are hampered by not having a definite test for the rheumatic state.

For the purposes of this survey chorea and growing pains as well as acute rheumatic fever have been included as evidences of rheumatic activity. Some workers may query the inclusion of Sydenham's chorea stating that it is purely a "nervous" disease but discussion of this view will not be given here. The date of onset of rheumatic carditis is also difficult to determine. Cases having only one episode of acute rheumatic fever are in the minority. Often acute rheumatic fever is preceded by a history of growing pains, chorea, scarlet fever or repeated tonsilitis. A patient may have several attacks of acute rheumatic fever and yet the heart may not be affected after the first attack. For the purpose of this series it has been assumed that the heart is affected and endocarditis has occurred during the first manifestation of the rheumatic state, be it chorea, acute rheumatic fever or a history of growing pains.

The duration of the acute rheumatic state is again difficult to decide. A single acute attack is

/not.....

not so common and many of the cases give a history of repeated sore throats, growing pains, chorea and acute rheumatic fever, all over a period of years. One can only assume that the carditis is also active during this period.

Table XVI shows the relevant past history of the 295 cases.

TABLE XVI.

Relevant past History in 295 Cases of Rheumatic Heart Disease.

| Previous History. | | No. of Cases. | Per Cent of Total. | |
|---|---|---|---|---|
| Rheumatic State. | Acute Rheumatic Fever. | 185 | 63 | |
| | Chorea. | 24 | 8 | 75 |
| | Growing Pains. | 12 | 4 | |
| | Scarlet Fever. | 14 | 4 | 25 |
| | No History. | 60 | 21 | |
| TOTAL ..... | | 295 | 100 | |

From this Table it will be seen that roughly three-quarters of the cases give a rheumatic history and one quarter give no history at all even after close questioning. Even if scarlet fever is included as a cause of rheumatic carditis there is still over one fifth of proved rheumatic heart cases with no history of the "rheumatic state". Many of the cases with no rheumatic history give a history of repeated sore throats and scarletina. It has not

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been possible to determine whether the incidence of sore throats and scarlet fever is higher in the cases that develop rheumatic carditis than in the healthy population.

(b). DURATION OF THE RHEUMATIC INFECTION.

If we further classify the 221 cases with a rheumatic history (Table XVII) according to the estimated duration of the infection we find only 43 per cent give a history where a single acute episode occurs and is apparently cured in under one year, and in almost one fifth of the cases attacks of the rheumatic state recur over a period of ten years or more.

TABLE XVII.

The Period of Time over which Clinical Evidence of the Rheumatic State was apparent, or recurred in 221 Cases.

| Duration. | No. of Cases. | Per Cent of Total. |
|---|---|---|
| Under 1 year. | 96 | 43 |
| 2 | 19 | 9 |
| 3 | 14 | 6 |
| 4 | 14 | 6 |
| 5 | 8 | 4 |
| 6 | 9 | 4 |
| 7 | 10 | 5 |
| 8 | 8 | 4 |
| 9 | 2 | 1 |
| 9 years and over. | 41 | 18 |
| TOTAL ..... | 221 | 100 |

/The.....

The recurrences consist of repeated attacks of acute rheumatic fever, or rheumatic fever followed by chorea, growing pains followed by rheumatic fever etc. Whether repeated cardiac damage is being sustained during this period is another question which will be dealt with later.

Table XVIII gives the number of attacks of the 185 cases giving a history of acute rheumatic fever.

TABLE XVIII.

Number of Attacks of Acute Rheumatic Fever in 185 Cases.

| No. of Attacks. | No. of Cases. | Per Cent of Total. |
|---|---|---|
| 1 | 120 | 65 |
| 2 | 50 | 27 |
| 3 | 11 | 6 |
| 4 | 1 | 0.5 |
| 6 | 3 | 1.5 |
| TOTAL | 185 | 100 |

It can be seen that two-thirds had only one attack of acute rheumatic fever and that the remaining third had anything between 2 and 6 attacks.

A further analysis of cases giving a history of acute rheumatic fever was carried out and is shown in Table XIX.

It can be seen that in relatively few cases

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(only 16 per cent) was acute rheumatic fever the only evidence of the rheumatic state.

TABLE XIX.

Associated Histories in 162 Cases having had an Attack of Acute Rheumatic Fever. Only live Cases are included as full Histories were thus obtained.

| History. | No. of Cases. | Per Cent of Total. | Relationship to the Attack. |
|---|---|---|---|
| Alone. | 25 | 16 | - |
| Plus Chorea. | 31 | 19 | 10 at same time.<br>4 before.<br>17 after. |
| " Growing Pains. | 15 | 9 | All before Rheumatic Fever. |
| " Scarlet Fever. | 37 | 23 | 28 long before.<br>8 just preceding.<br>1 just after. |
| " Repeated Sore Throats. | 16 | 10 | All before Rheumatic Fever. |
| " Erythema Nodosum. | 2 | 1 | Associated with Rheumatic Fever. |
| " Subsequent Rheumatism. | 36 | 22 | Subsequent Fibrositic Pains. |
| TOTAL .... | 162 | 100 | |

19 per cent also had chorea mostly at some time after the attack of rheumatic fever. It is interesting to note that 10 cases had rheumatic fever and chorea more or less at the same time. 9 per cent of the cases gave a history of growing pains before the attack of acute rheumatism. Scarlet fever and repeated sore throats occurred in 33 per cent of the cases almost universally before the acute attack.

Erythema nodosum was associated with the

/acute.....

acute rheumatism in 1 per cent of the cases. Following rheumatic fever, 22 per cent of cases complained of subsequent rheumatism over a period of years.

Although this Table does not show it, many of the cases had a combined history of scarlet fever, growing pains, acute rheumatic fever and chorea within a few years.

(c). AGE AT ONSET OF RHEUMATIC INFECTION.

Taking the first rheumatic manifestation as the age of onset we find from Table XX that there is a peak incidence around the age of 10 to 14 and over 50 per cent of all rheumatic infections start between the age of 5 and 14 years.

TABLE XX.

Percentage Distribution of Age at Onset of First Manifestation of the Rheumatic State. The Ages are grouped into five-year Periods.

| Age Group. | -5 | 5-9 | 10-14 | 15-19 | 20-24 | 25-29 | 30-35 | 35 |
|---|---|---|---|---|---|---|---|---|
| Percentage. | 0 | 25.0 | 38.3 | 20.1 | 12.4 | 2.3 | 1.4 | 0.5 |

There is a gradual fall in incidence between the ages of 15 and 24 and thereafter the incidence remains extremely low. In this series there was no case where the onset occurred before the age of 5 but between 5 and 10 years there is a rapid increase. This distribution is similar to that shown by Cohn and Lingg (1943a) but in their series of cases (including males and females) the maximum incidence

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fell at 8 years whereas here it is a year or two later. Their series however included cases dying in childhood whereas here only cases surviving to the childbearing age are studied. It can be seen that children are most liable to be affected with the rheumatic infection during the period of rapid growth - 10 to 16 years - and that during the childbearing age, rheumatic infection as an original occurrence is very rare, although a few cases may occur at the beginning of the childbearing period (18 to 24).

(d). RELATIONSHIP BETWEEN HISTORY OF RHEUMATIC INFECTION AND ULTIMATE FUNCTIONAL CAPACITY.

It has always been difficult to know the ultimate fate of cases developing rheumatic carditis during an attack of rheumatic fever. One is apt to assume that the prognosis in cases that develop severe carditis (including pericarditis), is less favourable than in cases where only a soft systolic murmur has been noted. Unfortunately in this series it has been impossible to obtain details about the actual severity of the original infection and the degree of cardiac involvement at the time of the acute episode. Some of the patients were able to say that they had pericarditis etc., but the evidence is insufficient.

However, the series can be divided into two groups, those with a rheumatic history and those

/with.....

with no rheumatic history. An attempt has been made to compare these groups with respect to their ultimate functional capacity according to their age groups. To do this correctly one must assume that the onset of the rheumatic carditis in those cases with no rheumatic history has occurred on the average at the same time as in the other groups. This is probably a reasonable assumption. All but 5 of the cases of rheumatic heart disease on the date of examination were between the ages of 20 and 50. Table XXI shows this relationship.

TABLE XXI.

Relationship between Functional Capacity at Different Age Groups and History of Rheumatic Infection in 290 cases.

| Age Group. | Rheumatic History. | No. of Cases. | Percentage of Cases. | | |
|---|---|---|---|---|---|
| | | | Grades 1 & IIa. | Grades IIb & III. | Dead. |
| 20-30 | Pos. | 70 | 77 | 14 | 9 |
| | Neg. | 16 | 76 | 12 | 12 |
| 31-40 | Pos. | 109 | 60 | 27 | 13 |
| | Neg. | 41 | 61 | 24 | 15 |
| 41-50 | Pos. | 37 | 40 | 40 | 20 |
| | Neg. | 17 | 58 | 12 | 30 |

Although there are relatively fewer cases giving no rheumatic history, the results are very striking.

In the two age groups 20 to 30 and 31 to 40 there is no difference in the functional capacity and

/mortality.....

mortality rate, the percentage being almost identical.

In the age group 20 to 30 about 75 per cent are only slightly handicapped, 12 per cent are severely handicapped and 12 per cent are dead, whether a previous rheumatic history is present or not. In the age group 31 to 40, 60 per cent are slightly handicapped, 25 per cent are severely handicapped and about 15 per cent are dead.

In the age group 41 to 50 there is some variation in the percentages a rather higher percentage of cases being severely handicapped in those patients giving a rheumatic history. About 50 per cent of the cases are only slightly handicapped and between 20 and 30 per cent are dead.

The similarity between the two groups in the ages between 20 and 40 is remarkable. The slight variation in the age period 40 to 50 may be accounted for by the smaller number of cases falling within this age period.

As has been pointed out, it has been impossible further to divide the group with rheumatic history into degrees of severity but the clinical examination of several cases where pericarditis was known to have been present, showed no unusual finding either in functional capacity or valvular defects compared with cases giving only a history of growing pains or mild rheumatic fever.

This study shows that the type of initial

/rheumatic.....

rheumatic infection has no influence in determining the eventual degree of cardiac damage, and that cases with no rheumatic history are in no way different from those that give a history. This statement does not include cases dying during the acute illness where a severe pancarditis may be fatal, but includes all cases surviving to the childbearing age and fit enough to bear children.

(e). RELATIONSHIP BETWEEN TYPE OF ORIGINAL INFECTION AND TYPE OF VALVULAR LESION.

An attempt was next made to find out if there was any difference between the extent of the ultimate valvular damage and the severity of the original infection. The valvular lesions of the 295 examined cases were classified as mitral disease alone (mitral stenosis and mitral incompetence) and mitral plus aortic disease. Table XXII shows the number of cases and percentage distribution in each of these classes of valvular disease in cases giving a rheumatic history (rheumatic fever, chorea, growing pains) and with no rheumatic history. The small number of cases giving only a history of growing pains and chorea makes the findings statistically inadequate. However the cases giving a rheumatic history are similar. Roughly 75 per cent develop mitral stenosis as the sole valvular lesion and 25 per cent develop combined mitral and aortic disease. The cases giving no rheumatic history show a somewhat

/different.....

different distribution.

TABLE XXII.

Relationship between Valvular Damage and Previous History.

| History. | Total No. of Cases. | Mitral Disease alone. | | Mitral plus Aortic Disease. | |
|---|---|---|---|---|---|
| | | No. | Per Cent | No. | Per Cent. |
| Rheumatic fever. | 185 | 133 | 72 | 52 | 28 |
| Chorea. | 24 | 18 | 75 | 6 | 25 |
| Growing pains. | 12 | 9 | 75 | 3 | 25 |
| No Rheumatic History. | 74 | 62 | 83 | 12 | 17 |
| TOTAL ... | 295 | 222 | 75 | 73 | 25 |

Far more (83 per cent) develop pure mitral stenosis as the sole lesion and far fewer (about 17 per cent) have combined mitral and aortic disease. Because of the relatively small number of cases giving no rheumatic history, these findings cannot be compared statistically with the group giving a rheumatic history. It would appear however that on the whole the type of initial infection does not influence the type of valvular lesion ultimately developing.

To sum up this section on the rheumatic infection, it is found that only three-quarters of the cases with rheumatic heart disease give a history of previous rheumatic infection. The age of onset

/of.....

of this rheumatic infection varies somewhat but occurs by far most frequently between the ages of 8 and 14 and it is rare as a first infection after the age of 22. The infection may occur as one episode lasting only a few months, but more commonly there are repeated episodes recurring over a period of years. The episodes may take a different form in any one case (acute rheumatic fever, chorea, growing pains). The form and severity of the initial infection do not influence ultimate valvular lesion or functional capacity of the heart nor does an absence of rheumatic infection influence markedly the ultimate cardiac damage. Histories of scarlet fever and repeated tonsillitis are common in cases which develop rheumatic heart disease, even when no other evidence of rheumatic infection is found. This finding tends to confirm the view that the rheumatic state is due to an altered response on the part of the patient to the streptococcus. It has been impossible to determine whether the incidence of scarlet fever and tonsillitis is greater in cases developing rheumatic heart disease than in the healthy population.

A further discussion on the rheumatic state will be given later.

(f). CLINICAL FINDINGS IN THE GROUP OF CASES EXAMINED.

Nutrition. The nutrition of the group as a whole showed no difference from the general population

/except.....

except in the severely handicapped or advanced cases, many showing auricular fibrillation, where nutrition was poor and even cardiac cachexia was present. A few cases also were markedly obese. An adenoid type of facies was common.

Cyanosis. A slight degree of cyanosis was seen only in the severely handicapped cases. The "mitral facies" was uncommon but occurred in a few of the advanced cases, especially those with poor nutrition. There was only one case with paralysis of left recurrent laryngeal nerve.

Clubbing of the Fingers. This was absent in all the cases except one which had developed subacute bacterial endocarditis.

Cardiac Rhythm. Normal sinus rhythm was present in 241 cases and extrasystoles detected in only 10 of those. 2 cases gave histories suggesting paroxysmal tachycardia and 13 cases had auricular fibrillation.

Blood Pressure. The blood pressure readings have been divided into readings on cases with mitral disease alone and readings on mitral plus aortic lesions. It should be pointed out that the readings were obtained after the patients had been recumbent for only a few minutes and are therefore "casual" blood pressure readings.

(a) Mitral Lesions. The average systolic pressure was 126 with a variation of ±14 and the average

/diastolic.....

diastolic pressure was 77 with a variation of ±10.

(b) Mitral plus Aortic Lesion. The average systolic pressure was 130 with a variation of ±20 and the average diastolic 66 with a variation of ±13. If we take the average blood pressure readings of the healthy population to be 122/72 ± 10 per cent we find that the cases with mitral lesion alone have blood pressures that fall within the normal range although a few have a higher systolic pressure and that the cases with an additional aortic lesion have a wider pulse pressure due to increased systolic and decreased diastolic pressures. It is perhaps worth noting at this point that the intensity of the aortic diastolic murmur was in no way a guide to the diastolic reading. Many cases that would have been judged to have free aortic regurgitation on auscultation had a diastolic pressure between 70 and 80.

Type of Lesion. In Table XXIII is shown the distribution of the valvular lesions. Of the 295 cases 222 or 75 per cent had mitral disease alone and 69 or 23 per cent had associated aortic disease. 4 had aortic disease alone (2 predominantly stenotic and 2 incompetent). Of the 222 cases with mitral disease alone 196 had definite stenosis with diastolic murmurs and 26 were classed as having predominant mitral incompetence where no definite diastolic murmur was heard. A diastolic mitral thrill was present in a large number of the cases of

/mitral.....

mitral stenosis.

TABLE XXIII.

The Distribution of the Valvular Lesions in 295 Cases of Rheumatic Heart Disease. The 222 Cases of Mitral Disease alone comprise 196 of pure Mitral Stenosis and 26 of Mitral Incompetence.

| Valvular Lesion | No. | Per Cent. |
|---|---|---|
| Mitral alone. | 222 | 75 |
| Mitral plus Aortic. | 69 | 23 |
| Aortic alone. | 4 | 2 |
| TOTAL ..... | 295 | 100 |

(g). RELATIONSHIP BETWEEN THE TYPE OF LESION AND THE FUNCTIONAL CAPACITY OF THE CASES.

On examination all the cases were classified according to their functional ability using the same method as is used during pregnancy. Table XXIV shows the results.

TABLE XXIV.

The Functional Grade of 254 Cases of Rheumatic Heart Disease on Follow-up Examination and the Average Age of each Grade.

| Grade. | Total No. | Per Cent of Total. | Average Age. |
|---|---|---|---|
| I | 16 | 6 | 31 |
| IIa | 163 | 64 | 35 |
| IIb | 63 | 25 | 37 |
| III | 12 | 5 | 41 |

It was found that 16 (6 per cent) had no handicap and they were classified grade I, 163 (64 per cent) were

/moderately.....

moderately handicapped and were graded IIa, 63 (25 per cent) were severely handicapped and graded IIb while 12 (5 per cent) were crippled and bedridden and graded III. Each functional grade was again divided into cases with mitral lesion alone and cases with mitral plus aortic lesions. Table XXV shows these figures with, in addition, the type of lesion present in the 41 cases which had died.

TABLE XXV.

Relationship between Functional Grade and Valvular Lesion.

| Grade. | Mitral alone. | Mitral plus Aortic. |
|---|---|---|
| | Per Cent. | Per Cent. |
| I | 80 | 20 |
| IIa | 77 | 23 |
| IIb | 61 | 39 |
| III | 77 | 23 |
| Deaths. | 83 | 17 |

It will be seen that there is a remarkable similarity in the percentage number of cases where mitral disease was the sole lesion - around 80 per cent in all grades with the exception of grade IIb where there is a lower incidence of cases with mitral involvement alone and a corresponding higher incidence of associated aortic disease. The reason for this is not apparent but one explanation can be offered.

/These.....

These grade IIb cases were advanced cases of rheumatic heart disease, with large hearts, and were bordering on pulmonary and peripheral oedema. Most were examined as out-patients after having walked a considerable distance and were still somewhat dyspnoeic. Diastolic murmurs were heard down the left side of the sternum which have been attributed to an aortic leak but it is possible that these diastolic murmurs in some cases were due to temporary pulmonary incompetence - Graham-Steele murmurs - and that they may have disappeared on further rest. The possibility that some of the diastolic murmurs were pulmonary in origin was further strengthened by the X-ray appearances which in some cases where aortic disease was diagnosed on auscultation, showed no radiological evidence of aortic disease - showing very small aortas and no left ventricular enlargement - but in those very cases there was unusual dilatation of the pulmonary conus and artery. Slight degrees of aortic leak show no peripheral signs which are typical of free aortic regurgitation and the second aortic sound may be pure even though a diastolic murmur is heard to the left of the sternum in the third or fourth interspace.

It is suggested then that the apparently increased incidence of aortic disease in the grade IIb cases is probably due to the inclusion of several cases where a Graham-Steele murmur of pulmonary

/incompetence.....

incompetence was heard due to temporary increase in the pulmonary blood pressure with dilatation of the pulmonary artery. Otherwise the figures show that whatever the functional grade of the group of patients the incidence of mitral and mitral plus aortic disease is the same. The converse also holds true - namely that the association of aortic with mitral disease occurs with equal incidence in all functional grades.

(h). RELATIONSHIP BETWEEN THE FUNCTIONAL GRADES IN TWO CONSECUTIVE PREGNANCIES.

As the state of health of a woman in a previous pregnancy is used as a guide to the prognosis on any subsequent pregnancy the analysis of the change in grade between the last pregnancy (x + 1) and the preceding pregnancy (x) was made and it was possible to compare the grades of two consecutive pregnancies. Table XXVI shows the results.

TABLE XXVI.

The Influence of Functional Grade in one Pregnancy (x) on the Functional Grade of a succeeding Pregnancy (x + 1).

| | | Functional Grade in Pregnancy (x). Percentage Distribution. | | | |
|---|---|---|---|---|---|
| | | I | IIa | IIb | III |
| Functional Grade in Pregnancy (x+1). | 1 | 15 | 1 | 0 | 0 |
| | IIa | 50 | 52 | 0 | 0 |
| | IIb | 25 | 31 | 65 | 14 |
| | III | 10 | 16 | 35 | 86 |

/If.....

If we take those cases who were grade I in one pregnancy (x) and find out their grade in the subsequent pregnancy (x + 1) we find that 15 per cent remained in grade I, 50 per cent were graded IIa, 25 per cent dropped into grade IIb and 10 per cent developed congestive heart failure - grade III. These cases who became grade III all had a period of over 10 years between the two pregnancies under consideration and the cases who became grade IIb all had an interval of over 5 years.

If we take the cases who were grade IIa in one pregnancy we find that in a subsequent pregnancy only one case (1 per cent) was better and regraded I. In the first pregnancy under consideration this patient had been convalescing from acute rheumatic fever but in the subsequent pregnancy she felt perfectly fit. 52 per cent were in the same grade in pregnancy x + 1, 31 per cent were grade IIb and 16 per cent were grade III.

Of the cases who were grade IIb in one pregnancy, not one was in a better grade in a subsequent pregnancy, 65 per cent were in the same grade and 35 per cent had become grade III.

Of the cases who were in grade III in a pregnancy and who became pregnant again, none fell into grades I or IIa. One case (14 per cent) was IIb at three months and the pregnancy terminated and 85 per cent were again in congestive failure.

/The.....

The small number of patients in this last group is accounted for by the fact that a patient once in congestive failure during pregnancy was usually sterilised and consequently a subsequent pregnancy did not occur. Only 7 cases, for one reason or another, had two pregnancies where severe congestive heart failure had developed. It should be pointed out that the x + 1 pregnancy in all cases was the last pregnancy that had occurred at the time of re-examination.

From these figures it is obvious that whatever a woman's health in one pregnancy she will never be in better health in a subsequent pregnancy. Grade I cases may develop severe symptoms in a subsequent pregnancy but this is due to a long interval between pregnancies and increased age of patient. It can be seen that more than half of the grade IIa cases remain in this grade in a subsequent pregnancy and under half are much worse - some developing congestive heart failure.

In the foregoing paragraphs a woman's grade in her last pregnancy has been compared to her grade in the immediately preceding pregnancy. However as many of the women had more than two pregnancies it has been possible to work out statistically the relationship between grades in successive pregnancies.

The units of information are pairs of successive pregnancies. Obviously a primaparous

/woman.....

woman affords no evidence on how grades in successive pregnancies are related. A woman who has had two pregnancies offers one piece of information, while a woman with ten pregnancies theoretically gives nine items. From the records of the multiparae it was possible to collect information relating to 460 pairs of consecutive pregnancies and these are classified in age groups in Tables XXVII (a), (b), (c) and (d).

TABLES XXVII (a), (b), (c) & (d).

Grade by Age at Pregnancy and Grade at Previous Pregnancy.

(a) Grade I at Previous Pregnancy.

| Age. | Grade at Subsequent Pregnancy. | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | Numbers. | | | | | Percentages. | | | |
| | I | IIa | IIb | III | Total | I | IIa | IIb | III |
| 18- | 26 | 3 | - | 1 | 30 | 87 | 10 | - | 3 |
| 24- | 74 | 14 | 6 | 1 | 95 | 78 | 15 | 6 | 1 |
| 30- | 33 | 23 | 6 | - | 62 | 53 | 37 | 10 | - |
| 36- | 6 | 10 | 11 | 4 | 31) | 18 | 35 | 35 | 12 |
| 42- | - | 2 | 1 | - | 3) | | | | |
| TOTAL | 139 | 52 | 24 | 6 | 221 | 63 | 23 | 11 | 3 |

/(b).....

(b). Grade IIa at Previous Pregnancy.

| Age. | Grade at Subsequent Pregnancy. | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | Numbers. | | | | | Percentages. | | | |
| | I | IIa | IIb | III | Total. | I | IIa | IIb | III |
| 18- | - | 12 | 3 | 2 | 17) | 1 | 72 | 19 | 8 |
| 24- | 1 | 54 | 14 | 5 | 74) | | | | |
| 30- | - | 41 | 16 | 7 | 64 | - | 64 | 25 | 11 |
| 36- | 1 | 15 | 13 | 7 | 36) | 2 | 42 | 32 | 24 |
| 42- | - | 2 | - | 3 | 5) | | | | |
| TOTAL | 2 | 124 | 46 | 24 | 196 | 1 | 63 | 24 | 12 |

(c). Grade IIb at Previous Pregnancy.

| Age. | Grade at Subsequent Pregnancy. | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | Numbers. | | | | | Percentages. | | | |
| | I | IIa | IIb | III | Total. | I | IIa | IIb | III |
| 18- | - | - | 2 | I | 3 | | | | |
| 24- | - | - | 7 | 2 | 9 | | | | |
| 30- | - | - | 10 | 2 | 12 | | | | |
| 36- | - | - | 7 | 2 | 9 | | | | |
| TOTAL | - | - | 26 | 7 | 33 | - | - | 79 | 21 |

/(d).....

(d). Grade III at Previous Pregnancy.

| Age. | Grade at Subsequent Pregnancy. Numbers. I | IIa | IIb | III | Total. | Total All Grades. |
|---|---|---|---|---|---|---|
| 18- | - | - | - | 1 | 1 | 51 |
| 24- | - | - | 1 | 2 | 3 | 181 |
| 30- | - | - | - | 2 | 2 | 140 |
| 36- | - | - | 1 | 3 | 4 | 80 |
| 42- | - | - | - | - | - | 8 |
| TOTAL | - | - | 2 | 8 | 10 | 460 |

In each case the functional grade at the latter pregnancy of the pair is shown in conjunction with the woman's age at that pregnancy and her grade at the previous pregnancy, the ages being combined into six-year groups. Actual numbers and percentages are given. Thus, of the 30 women who were grade I at a given pregnancy and then had another pregnancy between the ages of 18 and 23 inclusive, 26 were grade I at the latter pregnancy, 3 were grade IIa and 1 was grade III.

If an attempt is made to predict a woman's grade in a pregnancy from previous obstetrical history, there are many possibly significant variables such as age, parity, grade at each previous pregnancy, intervals between pregnancies and so on. By means of

/numerous.....

numerous tabulations, regression equations and tests of homogeneity which have been carried out by the statistician, it was established that, within the limits of significance possible with the number of cases dealt with:-

(a). A woman's cardiac grade at a given pregnancy is highly correlated with her grade at the immediately preceding pregnancy.

(b). The prognostic indications of grade at immediately preceding pregnancy vary with the woman's age.

(c). When age and grade at immediately preceding pregnancy are taken into account, the prognostic indications are not influenced by parity number or by grade at an earlier pregnancy. These earlier pregnancies can therefore be safely left out of account.

(d). Provided the interval between the last two pregnancies is not more than four years or thereabouts, the length of the interval does not appreciably affect the prognosis. For longer intervals the data are too sparse to give a significant answer, but there are indications that after a very long interval, a woman's heart at a subsequent pregnancy will tend to have deteriorated more than would be expected from the results given in the Tables.

In addition the Tables show that a woman's functional grade at a later pregnancy will almost

/always.....

always be as bad as, or worse than, her grade at an earlier pregnancy. In the 460 pairs of consecutive pregnancies studied, there are only 4 cases in which there was apparent improvement at the later pregnancy, and that never more than by one grade. Considering the women who were grade I at the earlier pregnancy, one finds that the probability of deterioration at the subsequent pregnancy increases strikingly with age. In the youngest group about 87 per cent showed no worsening compared with only about 18 per cent among those aged 36 or more. The likelihood that a woman who is grade I at a given pregnancy will drop to grade IIb or worse at her next pregnancy rises from about 3 per cent for the under 24 group to about 10 per cent for those 30-35 and nearly 50 per cent for those 36 and over. Similarly, when a woman is grade IIa at a given pregnancy is considered, her chance of being grade IIb or worse at her next pregnancy is about 1 in 4 if she is below 30, and more than 1 in 2 if she is over 36. There are relatively few cases in which a woman who was grade IIb or III at a pregnancy went on to have another pregnancy, since most of them were sterilised or advised against further pregnancy.

In summary then, and in so far as they go, the data indicate that about 4 women out of every 5 retain their grade in a subsequent pregnancy and the rest change for the worse by usually one grade only, and that this proportion does not vary with age.

/However.....

However the chances of a woman being worse off in a subsequent pregnancy are influenced by the length of time since her last pregnancy and by her age.

(i). FATE OF CASES: PROGNOSIS FOLLOWING PREGNANCY.

Of the 295 cases traced, 41 were found to have died anything between one and ten years following their pregnancy. The remaining 254 cases were in varying degrees of health. The Registrar-General for Scotland was good enough to help in tracing all the 41 fatal cases and the cause of death was ascertained in all. Table XXVIII shows the various main causes of death with the number and percentage in each group and also the average age.

TABLE XXVIII.

Cause of Death in 41 Cases.

| Cause of Death. | No. of Cases. | Per Cent of Total. | Average Age. |
|---|---|---|---|
| Congestive Heart Failure. | 25 | 61 | 37 |
| Embolism. | 6 | 14 | 39 |
| Sudden. | 4 | 10 | 32 |
| Subacute Bact. Endocarditis. | 2 | 5 | 33 |
| Subsequent Pregnancy. | 3 | 7.5 | 27 |
| Non-cardiac. | 1 | 2.5 | 22 |
| TOTAL ..... | 41 | 100. | |

It was found that 3 cases had died in congestive heart failure in a subsequent pregnancy

/during.....

during which no hospital ante-natal care had been obtained. 25 cases or 61 per cent of the total had died of congestive heart failure. The average age of this group was 36.7 years. All but 7 of these 25 cases had been in congestive heart failure during their last pregnancy. 5 cases had associated pulmonary embolic phenomena, 4 had associated respiratory infections and 1 had an attack of acute rheumatic fever.

The next largest group of 6 cases or 14 per cent of the total died as a result of embolism, in all cases cerebral embolism. One case had in addition emboli in the femoral arteries and another had a massive embolus which had lodged at the bifurcation of the aorta. Auricular fibrillation was known for certain to have been present in 4 of these cases. The average age of this group was 39 years.

4 cases (10 per cent) were noted to have died suddenly without any further cause being given. One of these deaths occurred in a case of aortic stenosis and may possibly have been due to acute left ventricular failure. The other 3 cases presumably died as a result of embolism. The average age of this group was 32 years. 2 cases died as a result of subacute bacterial endocarditis and 1 had in addition cerebral embolism. The average age of these 2 cases was 33 years. One case (2.5 per cent) died a

/non-cardiac.....

non-cardiac death, namely from spontaneous pneumothorax.

While the total number of cases is too small to analyse statistically it is obvious that the great majority of cases have died in congestive heart failure and that pulmonary embolism was an additional complication in a few of these.

On the other hand cerebral embolism as a main cause of death was usually not associated with any severe degree of congestive heart failure. Because of the relatively small numbers, little importance can be attached to the average ages of the different groups. It should be pointed out of course that most of the cases died at home and that the cause of death in these cases has not been proved at autopsy. All the deaths in the series occurred in cases that had been considered to have rheumatic heart disease during pregnancy.

The 254 women who were still alive at the time of the examination were in varying degrees of functional incapacity. Their past histories, clinical findings and functional gradings have already been studied. It remains now to study them in relation to their cardiac grades in their last pregnancy and to their grades on re-examination. All the available data were submitted to statistical analysis and it became evident that the most clear-cut results, as well as those of the greatest

/clinical.....

clinical interest and value, were those related to questions of prognosis. One of the most important tasks was to discover which items of information - patient's age, parity and so on - were essential for prognosis and which were relatively unimportant. Fortunately the practical conclusion turned out to be simple. To secure the fullest possible guidance in making a prognosis it was necessary to take into account only the woman's age, her functional grade in the last pregnancy and the period over which the prognosis was to extend. So far as the data under consideration went the prognosis was not significantly altered by taking into account also a woman's parity, her cardiac history or the state of her heart at pregnancies previous to the last one. The evidence for these conclusions is given below.

Table XXIX gives a summary classification of the patients whose records were analysed.

TABLE XXIX.

Findings at Follow-up Examination by Grade at last Pregnancy.

| | Grade at last Pregnancy. | | | | |
|---|---|---|---|---|---|
| | I | IIa | IIb | III | Total |
| Heart Disease on Follow-up. | 12 | 112 | 85 | 44 | 253 |
| Died before Follow-up. | - | 9 | 16 | 16 | 41 |
| Total. | - | 121 | 101 | 60 | 294 |
| Deaths per Cent. | - | 7.4 | 15.8 | 26.7 | |

/It.....

It will be seen that only 294 cases are included instead of 295 the reason being that one case was rejected from the statistical analysis as having insufficient information. The functional grades during the last pregnancy of all cases (those examined at follow-up examination and those who had died in the interval) are shown in the table.

All the deaths were certified to have died of heart disease, as previously shown, but it is possible, however, that certification may have been influenced by the fact that the women had attended the cardiac clinic during pregnancy. The crude death rates for grades IIa, IIb and III were respectively 7.4, 15.8 and 26.7 per cent.

It must be remembered that the present analysis omits to consider women who died during pregnancy or in the puerperium. The number of these (46) was considered to be too small to give significant answers to any questions about differential risks.

Knowing a woman's age and functional grade at pregnancy, what can one infer about her subsequent state of health and chances of survival? Information on this question is provided from the figures in Tables XXX (a), (b), (c) and (d). The primary grouping is by grade at last pregnancy. In each of these four major groups, the women are subclassified by age and grade at examination, or where the woman died before examination, by age at

/death.....

death.

## TABLES XXX (a), (b), (c) and (d).

Grade at last Pregnancy by Age and Grade at Examination or Age at Death.

(a). Grade I at last Pregnancy.

| Age. | Numbers. | | | | | | Percentages. | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | I | IIa | IIb | III | Dead. | Total. | I | IIa | IIb | III | Dead. |
| 24- | 3 | - | - | - | - | 3 | | | | | |
| 30- | 2 | 3 | - | - | - | 5 | | | | | |
| 36- | 1 | 2 | - | - | -) | ) 4 | | | | | |
| 42- | - | - | - | 1 | -) | | | | | | |
| Total. | 6 | 5 | 0 | 1 | 0 | 12 | 50 | 42 | 0 | 8 | 0 |
| Mean Age. | 31 | 35 | | 44 | | 33 | | | | | |

(b). Grade IIa at last Pregnancy.

| Age. | Numbers. | | | | | | Percentages. | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | I | IIa | IIb | III | Dead. | Total. | I | IIa | IIb | III | Dead. |
| 18- | - | 2 | - | - | 2 | 4) | | | | | |
| 24- | 2 | 31 | 2 | 1 | 2 | ) 38) | 5 | 78 | 5 | 2 | 10 |
| 30- | 4 | 28 | 1 | - | 2 | 35 | 11 | 80 | 3 | - | 6 |
| 36- | 1 | 27 | 2 | 2 | 2 | 34) | | | | | |
| 42- | - | 6 | 1 | 1 | 1 | ) 9) | 2 | 75 | 7 | 9 | 7 |
| 48- | - | - | - | 1 | - | ) 1) | | | | | |
| Total. | 7 | 94 | 6 | 5 | 9 | 121 | 6 | 78 | 5 | 4 | 7 |
| Mean Age. | 32 | 33 | 35 | 41 | 31 | 33 | | | | | |

/(c).....

(c). Grade IIb at last Pregnancy.

| | Numbers. | | | | | | Percentages. | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Age. | I | IIa | IIb | III | Dead. | Total. | I | IIa | IIb | III | Dead. |
| 18- | - | - | 1 | - | - | 1) | | | | | |
| 24- | - | 6 | 8 | - | 1 | 15) | 2 | 39 | 39 | 2 | 18 |
| 30- | 1 | 11 | 8 | 1 | 7 | 28) | | | | | |
| 36- | 1 | 17 | 13 | 1 | 3 | 35) | | | | | |
| 42- | - | 8 | 6 | 1 | 5 | 20) | 2 | 46 | 33 | 5 | 14 |
| 48- | - | 1 | - | 1 | - | 2) | | | | | |
| Total. | 2 | 43 | 36 | 4 | 16 | 101 | 2 | 42 | 36 | 4 | 16 |
| Mean Age. | 36 | 37 | 35 | 42 | 37 | 37 | | | | | |

(d). Grade III at last Pregnancy.

| | Numbers. | | | | | | Percentages. | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Age. | I | IIa | IIb | III | Dead. | Total. | I | IIa | IIb | III | Dead. |
| 24- | - | 5 | 4 | - | 3 | 12) | | | | | |
| 30- | - | 6 | 4 | - | 3 | 13) | - | 44 | 32 | - | 24 |
| 36- | 1 | 6 | 9 | - | 7 | 23 | | | | | |
| 42- | - | 3 | 2 | 2 | 3 | 10 | 3 | 26 | 37 | 6 | 28 |
| 48- | - | - | 2 | - | - | 2 | | | | | |
| Total. | 1 | 20 | 21 | 2 | 16 | 60 | 2 | 33 | 35 | 3 | 27 |
| Mean Age. | 40 | 35 | 36 | 43 | 36 | 36 | | | | | |
| Total All Grades. | 16 | 162 | 63 | 12 | 41 | 294. | | | | | |

/The.....

The total number of survivors and deaths in each main group must of course tally with the figures in Table XXIX.

Considering for the moment those who survived, it is apparent that in general the cardiac symptoms were less severe at the follow-up examination than they were during pregnancy. This is brought out by the following comparison of totals:-

| | I | IIa | IIb | III |
|---|---|---|---|---|
| Last Pregnancy..... | 12 | 112 | 85 | 44 |
| Follow-up Examination..... | 16 | 162 | 63 | 12. |

Although on the average the women were nearly four years older at the follow-up examination than at the last pregnancy the number of grades IIb and III fell from 129 to 75 a decline from 51 to 30 per cent of those examined. As the percentage analyses in Table XXX suggest and more refined statistical analysis confirms, age seems to make no significant difference to the distribution of grades at examinat: :ion among women of a given grade at the last pregnancy. The only exceptions are the 12 women classed as grade III at examination. Table XXX gives the average age in each sub-group. Women found to be grades I, IIa or IIb at examination did not differ significantly in age from the general average of their whole group. The grade III women, however, returned average ages of 44, 41, 42 and 43 years respectively, in each case

/several.....

several years above the general group average. The excesses considered together are statistically highly significant. It is also true that the mean interval between the last pregnancy and examination for the 12 grade III women was about 7 years against a general average of below 4 years.

If one considers survivors alone, the general picture that emerges is that pregnancy in a woman with heart disease greatly aggravates the usual symptoms; that within a few years after pregnancy the women in their twenties and thirties mostly show only slight impairment of functional efficiency, but that after a longer period, or in women in their forties, there is strong risk of complete invalidism.

These rather optimistic conclusions must be drastically modified when one also takes into account the numbers of women who died before they could be examined. As already noted in Table XXIX the crude death rate in the four main groups was respectively 0, 7, 16 and 27 per cent. In each group the age incidence of mortality was closely similar to that of survivors. A woman's risk of dying before examinat: :ion depended only on her grade at the last pregnancy, and not on her age. The number of deaths is too small to make it worth while applying life table methods or other elaborate statistical technique. It is sufficient to note that the average interval between the last pregnancy and death was just under 4

/years.....

years. In so far as the series of cases can be regarded as representative, one can infer that a woman who is grade IIa at pregnancy has a 1 in 14 chance of dying within a few years, a woman in grade IIb has a 1 in 6 chance, and a woman in grade III during pregnancy a risk of about 27 per cent. There is no reason to believe that these risks vary with a woman's age.

It is now possible to see how the data in Tables XXVII and XXX can be used for prognosis. Take for instance a woman aged 39 who was grade I at her last pregnancy. If she is again pregnant Table XXVII indicates that the risks of her dropping to grade IIa, IIb or III is respectively 35, 35 and 12 out of one hundred. Table XXX gives, for each eventuality, the risk that she will be dead in a few years. If one multiplies each grade risk by the corresponding death risk and adds the results, one obtains her total risk of dying within a few years of this future pregnancy. In the case under considerat: :ion the risk is 35/11 + 35/6 + 12/4 per cent which works out at about 11.3 per cent, or a 1 in 9 chance.

Table XXXI gives the precentage risks for different age and grade groups. As far as the data go, they support the wisdom of discouraging or preventing women who are grade IIb or III at pregnancy from undergoing any further maternal risk. They also raise the question of whether the same policy should

/not.....

not be extended to grade IIa women and to grade I women in their late thirties or forties.

TABLE XXXI.

Percentage Risk of Death after Further Pregnancy by Age and Grade at Previous Pregnancy.

| Age. | Grade at Previous Pregnancy. | | | |
|---|---|---|---|---|
| | I | IIa | IIb | III |
| 18- | 2 | 10 | 18 | 25 |
| 24- | 2 | | | |
| 30- | 5 | 12 | | |
| 36- | 11 | 15 | | |

In this connection one must take into account also the risk of death during pregnancy or childbirth. Figures already given show that this risk is around 2 per cent. The risk almost certainly varies with a woman's age and functional grade, but the number of deaths in Edinburgh was considered too small to allow of differential risks.

These quantitative conclusions based on the statistical analysis of the data, are subject to a formidable list of provisos and qualifications. The number of cases is too small to support any but the most tentative conclusions. The death rates are only rough and do not refer to any precisely defined period of risk. There is no similar data on the death risks of women with similar cardiac lesions who do not undergo pregnancy. It is possible that the

/extra.....

extra risk attributable to pregnancy is quite small.

In summary then the statistical findings confirm the clinical impressions with respect to prognosis both during and after pregnancy, namely that the functional grade is the most important single item in assessing prognosis in any given individual. Other factors, such as age of the patient, are of secondary importance.

The practice of terminating pregnancy and of preventing further pregnancy in any case that has become grade IIb or III in the past is fully justified by the statistical evidence.

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## 3. OTHER FORMS OF ORGANIC HEART DISEASE.

### (a). CONGENITAL.

On the follow-up examination nearly all the cases had rheumatic heart disease. The next biggest group of 10 cases or 3.6 per cent of the total number of organic heart disease cases had congenital flaws of various kinds. (Table XV)

The following is a list of the types of lesion encountered in this small series:

| | |
|---|---|
| Coarctation of the aorta | ..... 2 cases |
| Ventricular septal defect | ..... 2 cases |
| Auricular septal defect | ..... 2 cases |
| Patent ductus arteriosus | ..... 1 case |
| Subaortic stenosis | ..... 1 case |
| Pure pulmonary stenosis | ..... 1 case |
| Bundle branch block (left) | ..... 1 case. |

It is impossible to determine from the small

/number.....

number of cases the relative frequency of the different types of congenital flaw compatible with pregnancy. None of the above cases was severely handicapped during pregnancy and only one of the cases showed cyanosis (one of auricular septal defect).

As congenital heart disease is now more readily recognised and diagnosed it was thought that a study of a number of cases of congenital heart disease which had had one or more pregnancy would reveal some information as to the frequency of the types of lesion compatible with pregnancy.

The group comprised 38 cases of the following types:

| | |
|---|---|
| Patent ductus arteriosus ..... | 10 cases |
| Ventricular septal defect ..... | 6 cases |
| Auricular septal defect ..... | 5 cases |
| Pulmonary stenosis (single) ..... | 5 cases |
| Lütembacher ..... | 4 cases |
| Coarctation of aorta ..... | 4 cases |
| Subaortic stenosis ..... | 1 case |
| Complete heart block ..... | 1 case |
| Bundle branch block ..... | 1 case |
| Aortic septal defect ..... | 1 case. |

It can be seen that well over one quarter of all the cases have a patent ductus as the congenital lesion and that septal defects and pulmonary stenosis form over half of the remainder.

No attempt has been made in this thesis to go into further details about these congenital heart cases and how they fared during pregnancy, but it is considered that the group warrants further study and analysis.

/(b).....

(b). MISCELLANEOUS ORGANIC HEART DISEASES.

On follow-up examination (Table XV) 6 cases were found to have hypertensive heart disease. These 6 cases all had symptoms referable to the cardiovascular system during pregnancy and on re-examination these symptoms persisted. Obesity was a marked feature in 4 cases and in one of the others pre-eclamptic toxaemia had been a complicating factor during pregnancy. None of these cases had a valvular lesion. In all there was evidence of left ventricular enlargement on X-ray examination.

1 case had had thyrotoxic heart disease in pregnancy during which severe congestive failure had developed. She was eventually delivered through the pelvis but congestive heart failure persisted. This was controlled and subtotal thyroidectomy was performed. Thereafter she was much better and when seen four years later was leading a normal life, having had a normal pregnancy two years following the thyroidectomy. 1 case had suffered from syphilitic aortic disease with aortic incompetence during pregnancy without any cardiac embarrassment. She remained in good health when seen five years later.

No other type of organic heart disease was encountered in this series. It is noteworthy that no case of degenerative heart disease occurred. Fortunately the childbearing age and the age when degenerative heart disease is common do not coincide.

/Since.....

Since this article was commenced two cases have occurred. In one there was true effort angina in a woman of 35 during and after pregnancy and in the other coronary thrombosis with electrocardiographic changes developed 24 hours after labour. Her age was 34. There was also no case of pulmonary heart disease, though chronic bronchitis complicated many of the rheumatic heart disease cases.

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4. PATIENTS WHO ON FOLLOW-UP EXAMINATION HAD NO ORGANIC HEART DISEASE.

On follow-up examination 37 cases, or just over 10 per cent of the total, were found to have no organic heart disease (Table XIV). All these cases had been regarded as having organic heart disease during pregnancy, as they presented symptoms and signs of heart disease associated with varying degrees of functional incapacity. As it was rather surprising to find such a large number of cases with no cardiac lesion the group was investigated in more detail and classified as in Table XXXII.

It will be seen from this table that 2 cases were regarded as cardiac patients during pregnancy because of the presence of multiple extrasystoles associated with a history of mild exertional dyspnoea. On re-examination several years later both cases continued to show the cardiac irregularity without symptoms or signs of cardiac disease.

/The.....

The largest number of cases, 17 in all, were classified as cardiacs because of the presence of a mitral systolic murmur during pregnancy and they were all functionally graded IIa. 9 of them gave a history of rheumatic infection. On re-examination the murmur was still present in 12 cases but had disappeared in 5.

TABLE XXXII.

Distribution of 37 Cases found to have no Organic Heart Disease on Re-examination.

| Findings during Pregnancy. | No. | Rheumatic History. |
|---|---|---|
| Mitral Systolic Murmur. | 17 | 9 |
| Congestive Heart Failure. (Grades IIb and III). | 7 | 6 |
| Anaemia. | 5 | 3 |
| Extrasystoles. | 2 | 1 |
| Oedema due to Toxaemia. | 6 | 1 |

None of the patients were handicapped in any way and the cardiovascular system was negative on clinical, electrocardiographic and radiological examination. There was no relationship between a rheumatic history and the disappearance of the murmur.

6 cases had suffered from pre-eclamptic toxaemia and had been labelled cardiacs either because of left ventricular failure or because of suspected associated heart disease (rheumatic history). On follow-up no cardiac abnormality was detected.

/A.....

A further 5 cases had been considered cardiacs because of a mitral systolic murmur and marked functional disability falling into the IIb grade. They had all shown ankle oedema in association with exertional dyspnoea. In each anaemia had been a complicating factor during pregnancy but this had not been regarded as the primary cause of the disability. Table XXXIII shows the findings both during pregnancy and on follow-up examination.

It will be seen that none of the cases had lost their systolic murmur. 2 showed return of their haemoglobin levels to normal and both were symptomless. One of these had been sterilised after pregnancy as her condition (attributed to heart disease) had been so bad. The other had had two subsequent pregnancies without trouble. The other 3 cases still had evidence of anaemia in varying degrees with accompanying symptoms, but in all the heart showed no abnormality.

There remains a very interesting group of 7 cases all of whom had been severely ill during pregnancy and had shown signs of congestive heart failure, being classified grade IIb or III. Table XXXIV shows the findings during pregnancy and on re-examination. It will be seen that no patient in this group is under 30, the average age being high at 35.7 years, and all but one gave a history of rheumatic infection. During pregnancy none of these

TABLE XXXIII. The Findings During and after Pregnancy in 5 Anaemic Cases, all of whom were regarded as having Organic Heart Disease during Pregnancy.

| During Pregnancy. | | | | | | | On Re-examination. | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Age. | Parity. | Rheum. Hist. | Hb.% | B.P. | Grade. | Clinical Findings. | Age. | Grade. | B.P. | Hb.% | X-ray. | Clinical Findings. |
| 23 | 3 | Pos. | 47 | 104/60 | IIb | Mitral systolic murmur. | 33 | I | 118/70 | 90 | Neg. | Mitral systolic murmur. 2 subsequent Pregnancies. Grade I. |
| 40 | 5 | Neg. | 56 | 102/64 | IIb | " | 45 | IIa | 130/75 | 58 | " | Mitral systolic murmur. Anaemia. |
| 34 | 7 | Neg. | 60 | 118/60 | IIa | " | 42 | IIa | 120/80 | 65 | " | Mitral systolic murmur. Anaemia. |
| 42 | 11 | Pos. | 54 | 120/70 | IIa | " | 45 | IIb | 110/65 | 35 | " | Mitral systolic murmur. Anaemia. |
| 34 | 7 | Pos. | Low | 115/70 | IIa | " Pregnancy termin: :ated. Sterilised. | 41 | I | 132/85 | 102 | " | Mitral systolic murmur. No symptoms. |

TABLE XXXIV. The Findings during and after Pregnancy in 7 Cases regarded as severe Cardiacs during Pregnancy.

| During Pregnancy. | | | | | | On Re-examination. | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Age. | Parity. | Rheum. Hist. | B.P. | Grade. | Clinical Findings. | Age. | Grade. | B.P. | Hb.% | X-ray. | Clinical Findings. |
| 39 | 3 | Pos. | 120/70 | IIb | Mitral systolic Oedema of ankles. | 49 | I | 130/80 | - | Neg. | No murmur. |
| 42 | 2 | " | - | " | No murmur. Oedema. Digitalised. | 51 | " | 132/84 | 98 | " | No murmur. |
| 30 | 1 | " | 120/80 | " | Mitral damage. Oedema cleared on rest. | 33 | IIa | 136/80 | - | " | Soft Mitral systolic murmur. |
| 36 | 7 | Neg. | 100/64 | " | No murmur. Oedema cleared on rest. | 39 | " | 120/80 | 96 | " | No murmur. |
| 31 | 2 | Pos. | 115/70 | " | No murmur. Oedema cleared. Caes. Sect. Sterilisation. | 41 | I | 132/86 | 98 | " | No murmur. |
| 32 | 1 | " | 136/80 | III | M.S. in C.H.F. Caes. Sect. after digitilisation. | 33 | IIa | 120/75 | 102 | " | Soft Mitral systolic murmur. |
| 40 | 3 | " | 100/70 | " | No murmur. Leg and sacral oedema. | 47 | " | 120/70 | 94 | " | No murmur. |

patients had evidence of fever, anaemia, toxaemia and as far as can be judged their nutrition was within normal limits. None of them were hypertensive during pregnancy. On re-examination no cardiovascular abnormality was discovered clinically or radiologically. Haemoglobin values on 5 of the cases were normal and were not obtained on the other 2. Four of them still had mild exertional dyspnoea and could be graded IIa but the remainder were symptomless.

These 7 cases form a very remarkable group as they all showed what could be called congestive heart failure - oedema with dyspnoea and marked functional incapacity during pregnancy - and yet no demonstrable cardiac lesion existed. It is probably significant that all but one had a rheumatic history. It may well be that they suffered residual rheumatic myocardial damage without any valvular damage though on this supposition it is difficult to explain why they should be almost symptomless even ten years later. It may be that in certain women especially in the slightly older groups, the burden of pregnancy is too much for even a "normal" heart and actual cardiac decompensation occurs.

Another suggestion put forward is that for some reason or other excess fluid retention occurs during pregnancy and that all the tissues including the lungs, become oedematous.

Further discussion will be given in Part III.

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# PART III.

## DISCUSSION AND ANALYSIS OF THE FINDINGS IN PARTS I AND II.

### 1. INCIDENCE, TYPES AND MORTALITY RATES OF ORGANIC HEART DISEASE.

Over the past twenty years in the Simpson Pavilion the average incidence of heart disease in pregnant women is 1.33 per cent. Allowance must be made for this figure being too high, firstly because there is a tendency to refer cardiac cases to the Simpson thereby increasing the relative numbers, and secondly because roughly 10 per cent of the cases diagnosed during pregnancy as having heart disease have no detectable heart disease on follow-up examination. A figure of 1 per cent would probably be nearer the actual incidence of heart disease amongst pregnant women. Jensen (1938) believed 1 per cent to be the figure, basing his calculations on all the available data up to 1938. Authors report varying incidence figures from different centres. The variation is mainly due to the relatively larger number of cardiac cases dealt with in different hospitals, and probably partly due to an actual regional variation in the incidence of rheumatic heart disease which forms the majority of cases.

The types of heart disease encountered are few. Since the childbearing age occupies roughly the two decades 20 to 40, degenerative heart disease

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is very uncommonly met. Cases of coronary disease whether of myocardial ischaemia or infarction are a rarity during pregnancy and very few cases have been recorded. Likewise hypertensive heart disease is uncommon though hypertension may be more frequently encountered. Specific heart disease, rare anyhow in women is hardly ever encountered during pregnancy. Thyrotoxicosis is occasionally associated with pregnancy but like hypertension the heart is only rarely affected.

The vast majority of cases have rheumatic heart disease (about 94 per cent) and the next most important group is made up of various congenital flaws. Naturally the severer forms of congenital heart disease are not met with but there appears to be a fair number of types of congenital flaw compatible with pregnancy. The increased interest in this form of heart disease will probably lead to more diagnoses being made but at the moment our knowledge of the association of congenital heart disease and pregnancy is confined to a very few cases.

When discussing heart disease and pregnancy it is best to confine ourselves to rheumatic heart disease as it is only in this form of heart disease that sufficient numbers are available from which to draw conclusions. Our knowledge of the other forms of heart disease in association with pregnancy is

/relatively.....

relatively scanty and certainly from the small number of cases in this study no definite conclusions can be drawn.

During the twenty years under study the mortality rate has dropped considerably from over 6 per cent in the first 10 years to just over 2 per cent in the second 10 years. More instructive figures are probably obtained from the mortality rates in booked cases, that is those cases having had ante-natal care. These rates are 3.5 per cent and 1.7 per cent respectively. These rates are very slightly below the true mortality rate in heart disease as they are percentages of the total number of cases listed as cardiacs during pregnancy. It has been shown however that roughly 10 per cent of such listed cases have in fact no heart disease on "follow-up" examination. Even making allowance for this, the death rate in the past few years has been below 2 per cent. In order to give the mortality rates for rheumatic heart disease, a further slight correction is necessary involving less than 0.1 per cent, as about 5 per cent of the true cardiac cases have factors other than rheumatic fever as the aetiology. All the deaths however occurred in subjects of rheumatic heart disease. For practical purposes therefore the figures presented represent the mortality rates during pregnancy with regard to rheumatic heart disease. Similar reduction has

/occurred.....

occurred in other centres. Bunim and Rubricius (1948) who summarised all the available data in the ten year period 1936 to 1946 found an average mortality rate of 3.4 per cent out of 4,869 pregnancies in subjects of rheumatic heart disease compared with a figure of 9.38 per cent for the years 1890 to 1922. Improvement in these figures is still taking place but it is probable that it will be difficult to improve upon a mortality rate of 1 per cent even with the best ante-natal supervision.

Over the same period of years there has been a marked reduction in the total maternal mortality rate from all causes from 1.1 per cent down to 0.18 per cent due mainly to better therapeutic measures for cases of sepsis, haemorrhage and shock. No doubt cardiac cases have benefitted from these measures but the cardiac mortality rate has not fallen to such an extent. Haultain (1948) listing the main causes of maternal mortality in different centres, shows that twenty years ago cardiac disease was well down in the list, whereas nowadays it is one of the most important factors and indeed sometimes, the most important factor in the overall maternal mortality rate.

The reason for this is not difficult to find. New and beneficial therapeutic measures have been discovered and used to prevent death from such causes as sepsis, haehorrhage and shock. Sulphonamides,

/penicillin.....

penicillin and transfusion of blood or plasma have all contributed greatly in the past few years to the improvement in the death rate. In cardiology no such new therapeutic advance has been made. With the exception of the treatment of bacterial endocarditis, there has been little or no change in the treatment of rheumatic heart disease and its complications in the last ten years or more. Changes have taken place in the management and care of these patients, and have caused a marked reduction in mortality. Improvement has been gained more by prevention of complications rather than by advances in treatment of the complications of heart disease.

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2. CIRCULATORY ADJUSTMENTS IN PREGNANCY.

While it has been known for many years that the work of the heart is increased during pregnancy no really reliable method of measuring the output of the heart was available. With the advent of cardiac catheterisation however it is now possible to measure accurately the cardiac output. Several studies of the cardiac output in pregnant women have recently been done (Palmer and Walker, 1949 and Hamilton, 1949). These studies go to show that from the tenth week onwards there is a steady increase in the minute output reaching a maximum of 27 per cent above the

/non-pregnant.....

non-pregnant level about the 28th week. During the last 3 to 4 weeks of pregnancy there is a sudden fall in the output. This rapid decrease continues into the puerperium and a normal output is again reached at the end of this period. In the non-pregnant state the heart on the average has an output of 4.5 litres per minute under basal conditions. During pregnancy the output is increased to 5.7 litres per minute under similar conditions (Hamilton, 1949). There is probably more than one factor responsible for this increase in output but the many factors postulated do not interest us here and there is little point in discussing them. The important point is that the heart must increase its work by about 25 per cent during pregnancy up till the last month when there is a marked drop in cardiac output. As will be pointed out later this decrease in the amount of work the heart has to do is of practical importance.

Other cardiovascular changes of clinical importance may occur in normal women during pregnancy. Functional systolic murmurs are common both in the mitral and pulmonary areas (Gammeltoft, 1928) and there is often accentuation of the pulmonary sound. These may be increased venous pulsation in the neck.

It is now generally agreed that the normal heart does not hypertrophy during pregnancy but the pregnant uterus pushing up the diaphragm causes cardiac displacement. The heart is compressed from

/below.....

below upwards and lies in a more horizontal position with resultant electrocardiographic changes, and there is often backward displacement of the oesophagus by the left auricle.

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3. DIAGNOSIS OF HEART DISEASE DURING PREGNANCY.

As a result of the increased burden on the heart it is not surprising that most normal women experience exertional dyspnoea as pregnancy progresses. Often only slight dyspnoea is noticeable but sometimes quite marked respiratory embarrassment may be present. Many women present symptoms suggestive of heart disease and physical examination may reveal signs which closely mimic organic heart disease. A definite diagnosis of heart disease is therefore often very difficult to make during pregnancy. If definite signs of a valvular lesion are present no difficulty arises but even the characteristic murmurs of organic heart disease may vary in the course of pregnancy. In the series of cases studied here, roughly 10 per cent on follow-up examination were found to have no cardiovascular disease clinically and on radiological examination and yet during pregnancy these had all suffered various degrees of disability and were considered to be suffering from heart disease. The main causes for difficulty in diagnosis are as follows:

/(a).....

(a). MITRAL SYSTOLIC MURMUR.

The presence of a mitral systolic murmur even in the normal person may be difficult to interpret, especially if there is a history of rheumatic fever, and this difficulty is further increased during pregnancy when exertional dyspnoea is the rule. Naturally such cases are considered to have rheumatic valvular disease. About one third of the cases with a mitral systolic murmur during pregnancy showed no murmur on re-examination. The remainder continued to show a murmur, which from its character, could be classified as functional. Little or no help can be expected from other diagnostic aids. The electrocardiograph shows no diagnostic change in early rheumatic valvular disease. X-ray examination may be very misleading during pregnancy. Even during the early months the pulmonary artery may show more than normal fulness and the left auricle may appear slightly enlarged due to higher position of the diaphragm (Roesler, 1946). These changes become accentuated in the later months. Radiologically it is therefore almost impossible to diagnose with certainty the early changes due to mitral stenosis (Hollander and Crawford, 1943) and differentiate them from the changes that may occur during pregnancy. Gerhardt (from Jensen) states that during pregnancy the heart has a "mitral shape without mitral lesion".

/In.....

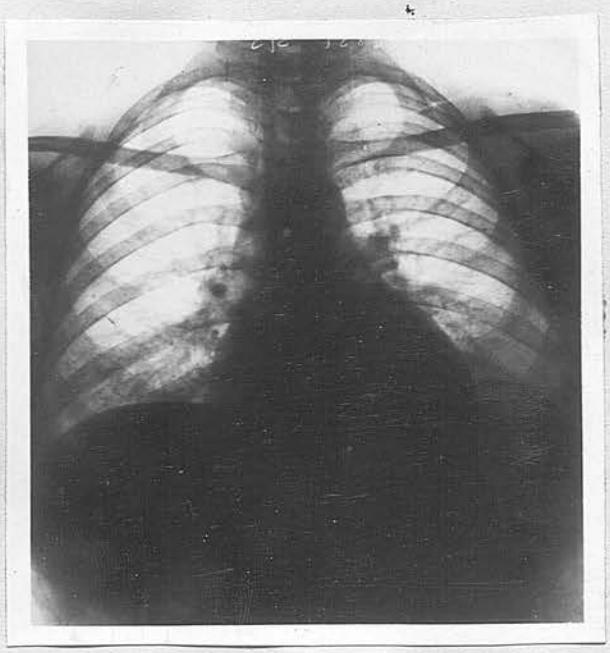
In Figure I are shown the X-ray findings in one of the cases from Table XXXIV. Both anterior and right oblique views are shown. During pregnancy this woman was considered to have rheumatic heart disease with congestive failure. The films taken about 10 weeks before term indicate how easy it was to confirm the diagnosis radiologically - there is enlargement of the left auricle with congestive changes in the lungs. The later films, taken about 1 year after delivery show a normal heart shadow.

Even in the non-pregnant state X-ray examination may not help in diagnosing cases of early mitral valvular disease. Those very cases that are doubtful clinically show also doubtful X-ray changes. The diagnosis therefore depends mainly on the character of the mitral murmur. In pregnancy then it is not surprising that many cases with a mitral murmur are considered to have a valvular lesion when none exists.

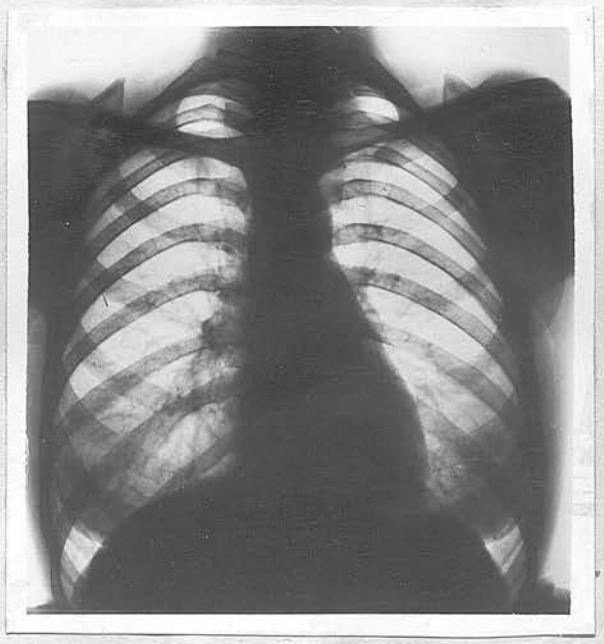
On follow-up examination a systolic murmur was considered organic if it was loud and associated with radiological changes, and such cases were considered to have rheumatic mitral incompetence. If the murmur was soft and associated with pure heart sounds and no radiological evidence of cardiac enlargement, it was considered to be functional, irrespective of a previous rheumatic history.

/Figure I .....

FIGURE I.



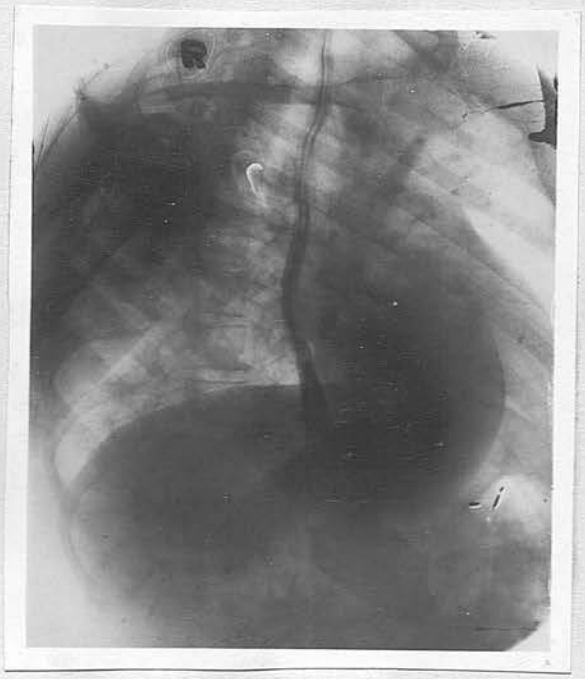
(a).



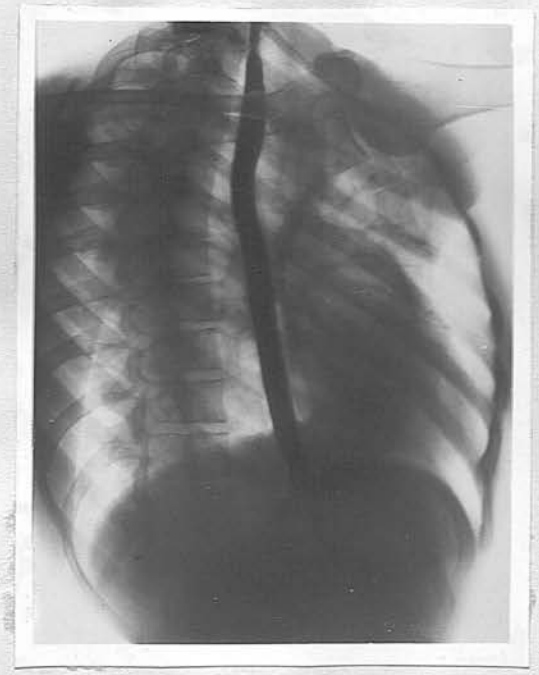
(b).

Anterior views of the heart of the same patient (a) about 10 weeks before delivery and (b) one year following delivery. Note the high diaphragm and apparent cardiac enlargement with pulmonary congestion in (a) and normal appearance of heart and lungs in (b).

FIGURE I (Contd.)



(a).



(b).

Right oblique views of the same patient taken at the same times as the anterior views. Note the definite backward displacement of the oesophagus by the left auricle in (a) simulating mitral stenosis, and the straight oesophagus in (b).

(b). ANAEMIA.

Many women during pregnancy develop an iron deficiency anaemia and complain of associated symptoms of dyspnoea, tiredness and occasionally of ankle oedema. Anaemia is also common in women suffering from rheumatic carditis (Jensen, 1938). In this series several women suffering from fairly marked degrees of anaemia were considered to have rheumatic heart disease. All were fairly severely handicapped (grades IIa and grades IIb) and all had a mitral systolic murmur. More than half of them gave a rheumatic history which may have lent support to the diagnosis of rheumatic heart disease.

On re-examination after pregnancy no cardiovascular disease could be detected and it must be concluded that their disability during pregnancy was entirely due to anaemia, which may closely mimic rheumatic heart disease.

(c). PRE-ECLAMPTIC TOXAEMIA.

A few cases in this series were considered to have heart disease associated with toxaemia of pregnancy. Patients with pre-eclamptic toxaemia often show signs suggestive of congestive heart failure - dyspnoea, peripheral and pulmonary oedema - and this in association with a mitral systolic murmur and a rheumatic history, may lead to a diagnosis of rheumatic heart disease, and may also account for the belief that pre-eclamptic toxaemia occurs more

/frequently.....

frequently in women with rheumatic heart disease (Carr and Hamilton, 1933), (Corwin and Herrick et al, 1927).

There is no doubt that the hypertension associated with the toxaemias may cause acute left ventricular failure which may prove fatal. Several such cases were included in the annual reports as having cardiac disease whereas strictly speaking they should have been classified under toxaemias of pregnancy.

(d). "FICTITIOUS HEART DISEASE".

Several cases had during pregnancy symptoms and signs of congestive heart failure, severe enough in some to require digitalis and mercurial diuretics, and yet no demonstrable heart lesion was found on re-examination several years later. None of these patients had evidence of toxaemia, anaemia, hypertension, sepsis or malnutrition which might account for their symptoms. It is perhaps significant that the age in every case was over 30 and probably more significant that every case but one gave a history of rheumatic infection. It may be that these patients suffered permanent myocardial damage during their rheumatic infection without at the same time receiving endocardial damage, and that consequently their myocardial reserve was insufficient to deal with the burdens of pregnancy. It has often been suspected that rheumatic fever may result in

/permanent.....

permanent myocardial damage in the absence of endocardial or pericardial damage. Post-mortem examination would be necessary to prove this point. If the myocardium were damaged one would expect some degree of functional incapacity to be present even when the patient was not pregnant. In this group of cases no such impairment was noticed following the pregnancy. Symptoms and signs cleared up rapidly and a normal cardiovascular system was found on examination.

It has long been known that pregnant women with apparently healthy hearts sometimes develop conditions which closely resemble organic heart disease (Handfield-Jones, 1890). Jensen, F.G.(1927) determined the incidence to be 39 cases out of a series of 239 apparently healthy women - a very high proportion. The etiology is obscure. There is obviously a disproportion between the cardiac reserve and the load placed on it by pregnancy. Whether the load is excessive or whether the reserve is naturally small or decreased by some factor is not known. Various theories have been postulated. Mild thyrotoxicosis has been suggested as a cause (Jensen, 1938). There may be decreased cardiac reserve, perhaps due to an inherently poor myocardium, perhaps due to residual damage following rheumatic fever, as the group in this series would suggest.

Recent work by Chesley (1944) has shown

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that part of the increase in weight experienced by all pregnant women is due to water retention. He considers that an increased rate of water retention, to such an extent that oedema becomes apparent, occurs as a manifestation of incipient toxaemia. The cause of the normal and abnormal water retention is not obvious, but may be due to the presence of large quantities of steroidal sex hormones which in their turn may be responsible for the sodium retention found in pregnancy. Most of the retained fluid is in the blood itself but a fair amount is in the intercellular tissues.

It may be then that these cases showing "cardiac" failure during pregnancy have an abnormal degree of water and sodium retention without other evidence of toxaemia, and that the lungs themselves become oedematous to a greater or less extent, causing dyspnoea and eventual cyanosis. These signs with the additional sign of peripheral oedema, can easily be considered due to congestive heart failure.

It is interesting to note that on the follow-up examination many women with heart disease who were bordering on failure stated that their symptoms of exertional dyspnoea and ankle oedema were worse premenstrually, at the time when the blood oestrogen is at its highest level and water and sodium retention likely to be increased.

The symptoms in pregnancy appear in persons

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who have previously shown no signs or symptoms of heart disease. They may come on early in pregnancy but as a rule they do not appear until the second half. The symptoms are those of gradually progressing heart failure: palpitation, dyspnoea, oedema at first of the ankles, later becoming more extensive, and finally orthopnoea and cyanosis. The physical signs mimic closely heart failure from organic heart disease - tendency to tachycardia, crepitations at lung bases and in advanced cases general anasarca. There are however no murmurs diagnostic of valvular damage and in some cases no murmurs are noted. Signs and symptoms clear up under treatment as for congestive heart failure - bed rest, digitalis and mercurial diuretics. The difficulty in distinguishing organic signs from functional signs during pregnancy has already been pointed out.

As is generally known, most women experience symptoms of cardiac embarrassment during pregnancy and it may be that this group exhibit a further stage. Some patients suffer merely from slight dyspnoea while others develop complete congestive failure. Most authors seem to think (and this view is confirmed in this series) that the prognosis is always good and that after delivery the changes clear up without permanent damage to the heart, but Jensen (1938) himself was not so decided on this point. He stated that in almost every published series of fatal cases

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of heart disease and pregnancy there are a few instances of death occurring from unspecified myocardial failure, which he considered might be due to the more serious forms of functional heart disease. Four cases of acute myocardial failure occurred in this series and post-mortem showed no definite signs of cardiac disease. None of these however had presented symptoms and signs of cardiac failure during pregnancy, death occurring suddenly during or after labour, so that they do not form a comparable group to the cases which develop congestive failure during pregnancy. We have had no such case dying during pregnancy or the puerperium.

Further study is necessary to determine the cause of failure in these women.

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4. THE VALUE OF FUNCTIONAL GRADING TO ASSESS PROGNOSIS DURING PREGNANCY.

Mackenzie (1921) was the first to emphasise the importance of the functional capacity of the heart in assessing prognosis. He wrote: "Estimation of the significance of murmurs, as of all other signs, should be based not on the murmur itself, but on the functional efficiency of the heart. When there is marked inefficiency of the heart, shown by breathlessness on slight exertion, rapid pulse, or easily excited palpitation, then there is danger in

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pregnancy". Since then all authors have agreed with Mackenzie's findings, but have differed in their assessment of the relative significance of other factors, such as age, degree of structural damage etc., in deciding prognosis. In Edinburgh the prognosis has been based almost entirely on the functional capacity and little attention paid to other factors. On the follow-up findings and on the statistical evidence it was possible to justify this attitude, and considerable study was given to the relationship between the functional grading and other clinical findings. For over 20 years the grading of the functional capacity of patients with heart disease has been done according to the criteria laid down by the New York Heart Association (1939) and later adopted by the British Cardiac Society. The Grades are as follows:-

Grade I. Patients with cardiac disease and no limitation of physical activity. Patients in this grade do not have symptoms of cardiac insufficiency nor do they experience effort pain. The presence of an organic lesion is usually discovered on routine examination.

Grade IIa. Patients with cardiac disease and slight limitation of physical activity. Ordinary physical activity causes undue fatigue, breathlessness or palpitation. They are usually fit to do all but heavy housework.

/Grade IIb.....

Grade IIb. Patients with cardiac disease and marked limitation of physical disability. They are comfortable at rest but slight activity causes undue fatigue, breathlessness and palpitation. They are on the verge of congestive failure and may show ankle oedema at night.

Grade III. Patients with cardiac disease who are unable to carry on any physical activity without discomfort. They show evidence of congestive heart failure.

By this means of grading it is possible to put every patient into one of four grades. If the methods and criteria for grading are carried out carefully there should be little chance of error due to the personal element on the part of the doctor. The following factors were studied with reference to their relationship to the functional grading.

(a). AGE: Generally speaking the older the patient the further down the functional scale she is. This applies both during pregnancy and in the non-pregnant state. This finding is really self-evident. Rheumatic heart disease runs a downward course, the rate of the downward trend varying in any given individual, so that the older patients tend to be more incapacitated than the younger. During pregnancy those who remain well compensated are under 30 years of age. Over the age

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of 30 there is a tendency for severe incapacity and congestive failure to develop. These are the general findings but it is difficult to apply this knowledge to any given individual. There is a wide variation in the rate of progression of rheumatic heart disease, some women of 20 developing congestive heart failure, and others living to over 40 years without developing this complication. There is a wide scatter in the ages at which congestive heart failure develops. In assessing a prognosis in an individual the age is therefore of little significance but it can be expected that over the age of 30 severer degrees of handicapping will occur during pregnancy.

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(b). DURATION OF RHEUMATIC INFECTION.

Because the age was not reliable in assessing prognosis many workers have considered that the length of time a patient has had heart disease might be a better indication (Bunim and Rubricius, 1948). This might be true if it were possible to know exactly when the heart had been damaged. As has been pointed out in Part II, many cases give no past history of rheumatic infection and of those that do only a few can give a definite date - most giving a vague history extending over years. It is therefore considered that from a practical point of view it is more or less impossible to assess prognosis

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on this basis. It has likewise been impossible to correlate the functional grading with the duration of the cardiac damage, on the information available in this series.

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(c). SEVERITY AND TYPE OF ORIGINAL RHEUMATIC INFECTION.

It has been found here that neither the severity of the initial rheumatic infection nor the number of attacks bear any relationship to the ultimate functional capacity. This statement includes only those cases surviving the repeated attacks and in whom the rheumatic state has probably become quiescent. There is no doubt that repeated attacks of rheumatic fever may further damage the heart and that death may occur during an attack especially in adults, but apparently a child who suffers several attacks of rheumatic fever over a period of years and survives them is ultimately no worse off than a child who develops endocarditis without a history of rheumatic fever.

The past history is therefore of no use in assessing prognosis during pregnancy and bears no relationship to the functional capacity at any given age.

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(d). TYPE OF VALVULAR LESION.

/No.....

No relationship could be found between the type and severity of the original infection and the type of lesion developed. Similarly, no difference in functional capacity could be made out between a single mitral lesion and combined mitral and aortic lesions, the percentage of single and combined lesions being the same in all the groups. This finding therefore agrees with those of Hamilton (1937), McIlroy and Rendel (1931) and De Graff and Lingg (1935). In assessing prognosis the type of valvular lesion is of no value. The main value of the valvular lesion is in diagnosis.

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(e). TYPE OF RHYTHM.

The main serious abnormality of rhythm encountered in rheumatic heart disease is auricular fibrillation. This has always been considered to carry a poor prognosis and the few cases who exhibited this arrhythmia on examination were all grade IIb or III (i.e., severely handicapped). It is seldom that a patient with fibrillation is well compensated and from past experience all patients with this irregularity do badly in pregnancy. The presence of extrasystoles (when not due to digitalis) appears to have little untoward effect.

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(f). HEART SIZE.

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Although it has not been possible to give definite proof from the series in this instance, it appears that generally speaking the higher the grade of functional incapacity the larger the heart. There are however many exceptions to this rule, some women with little or no handicap having large hearts, so that in an individual case the size of the heart per se is of little importance in assessing prognosis.

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From these findings the functional capacity of the heart is the one important factor in judging the ability of a woman to go through pregnancy with safety. The capacity bears no relationship to certain factors such as past history and type of valvular lesion, but generally speaking is closely related to age and heart size. Older age and larger hearts occur pari passu with a decrease in functional capacity.

Since the functional grading of an individual is of the greatest importance, much care must be taken in placing the patient in the correct grade. This can only be done by careful questioning on the ability to do daily work, climb stairs, play games etc. From experience gained over years in Edinburgh and every other centre using the grades as recommended by the New York Heart Association, patients who are grade I do well throughout pregnancy. They may fall into grade IIa in the later months but not lower.

/Patients.....

Patients who are grade IIa as a rule do well provided certain precautions are taken. A few may fall into grade IIb but with rest in bed they usually revert to grade IIa and they very seldom become grade III. Women who are grade IIb or III or who have ever been in that grade never do well if pregnancy is allowed to occur.

A better method of assessing prognosis in pregnancy is of course a previous test of pregnancy. In this series it was found that upgrading in a subsequent pregnancy hardly ever occurs. 4 out of 5 women do just as well and the rest are worse off in a subsequent pregnancy - they are never better. Also if congestive failure has developed during pregnancy it will develop again in a subsequent pregnancy. These findings confirm the findings of Bunim and Rubricius (1948). The series here also showed that a drop of more than one grade in a subsequent pregnancy is unusual but did occur if the time interval between pregnancies was long. For instance one or two cases grade I in one pregnancy were grade III in their next pregnancy, but an interval of over 10 years had occurred. However, provided the interval between the two pregnancies is not more than 4 years or thereabouts, the length of time does not appreciably affect the prognosis.

To summarise then, it is considered that the main factor in evaluating prognosis in pregnancy

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is the functional capacity of the heart.

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## 5. CARDIAC COMPLICATIONS LIABLE TO OCCUR DURING PREGNANCY.

As has been pointed out before, the output of the heart increases during pregnancy and consequently a fair amount of cardiac reserve is necessary to enable the extra work to be carried out efficiently. If the cardiac reserve is insufficient, congestive heart failure occurs.

### (a). CONGESTIVE HEART FAILURE.

This is by far the commonest complication of pregnancy, accounting for 61 per cent of the deaths in this series, and 57 per cent in Hamilton and Thomson's (1941) series, although the majority of women who develop this complication recover.

The incidence of severe congestive heart failure (grade III) was 64 out of 295 cases or 21.6 per cent. In the series studied by Bunim and Rubricius (1948) the incidence was 14 per cent. Jensen (1938) considered the incidence to be about 20 per cent (Carr and Hamilton 19.2 per cent, Jaschke 23.5 per cent, Fromme 21.4 per cent and Laennec 21.5 per cent, all quoted by Jensen). If cases who showed early signs of failure (grade IIb) are included the incidence becomes 156 out of 295 cases

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or 52.9 per cent of the total. It has been impossible from lack of definite information, to relate the incidence of congestive failure to the duration of pregnancy but the impression is that congestive failure, if not present previous to pregnancy occurring, seldom appears in the first two months but thereafter the incidence increases steadily till term, and that the appearance of congestive failure for the first time in the last months of pregnancy is rare. Hamilton and Thomson (1941) confirm these impressions and point out in addition that in their series of cases congestive heart failure seldom occurs for the first time at or following delivery. It appears then that the incidence of failure is directly related to the increase in cardiac work - increasing till the end of the eighth month, and diminishing during the last month.

However, the incidence of congestive failure and death differ markedly in relation to the phase of gestation. While decompensation in most cases occurs during pregnancy, death usually follows delivery. This fact is well borne out in Table VIII where out of 28 deaths due primarily to congestive heart failure 16 occurred after delivery, the remainder occurring equally in the first and second halves of pregnancy. This finding has been noted by several workers, especially Jensen (1938) who tabulated his

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results in a series of 462 fatal cases and found that only 15.7 per cent died during pregnancy, 9.4 per cent during labour and 43.9 per cent within one week of delivery the remainder dying from the second week to the fifteenth month following delivery. (Jensen however does not specifically state that congestive heart failure was the cause of death in his series). In his series 17 per cent, or as many as died during pregnancy, died during the first 24 hours following labour.

In this series 10 out of the 16 cases dying in the puerperium died within 24 hours of delivery (i.e., 35 per cent of all the deaths due to congestive heart failure occurred within 24 hours of delivery). We had no death from this cause during labour. All the available evidence goes to show that the few hours following delivery are the most dangerous for a woman in congestive failure. Such women may be safely nursed through pregnancy to meet a sudden death soon after labour. No entirely satisfactory reason for this has been given but it would appear that the circulatory mechanics are suddenly altered during or immediately following labour causing an overloading of the already weak heart. The most attractive hypothesis (Hoffman and Jeffers, 1942) is that a large amount of blood contained in the pregnant uterus is expelled when this organ contracts following delivery of the child, so causing a sudden increase in

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the volume of blood returned to the right heart, and that the already damaged heart is unable to cope with this additional load. There is as a result development of pulmonary oedema which proves fatal, analagous to overloading of the circulation during transfusion of fluids.

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(b). ACUTE PULMONARY OEDEMA.

Another important cause of death during pregnancy is acute pulmonary oedema in the absence of congestive heart failure. It is fortunately not common. In this series only 6 cases were encountered and they formed 13 per cent of all fatal cases. It has not been possible to give the incidence of this complication as only the fatal cases have been recorded. Of the six fatal cases one occurred during the second stage of labour, three occurred within a few hours of delivery, one in the first half of pregnancy and one in the second.

The precipitating cause of acute pulmonary oedema is not known. It is known to occur very rarely, in the non-pregnant state, in subjects with mitral stenosis and has been recognised as a complication in pregnancy for many years. Its onset is sudden and may be associated with a respiratory infection, especially a cough.

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In a series of cases studied by Jensen(1938) acute pulmonary oedema accounted for 10 per cent of all deaths in rheumatic heart disease complicated by pregnancy. He found that 22 per cent of these deaths occurred during labour and 29 per cent within 12 hours of delivery. Although the number of deaths in the Edinburgh series is too small, it is certainly possible to confirm Jensen's statement that pulmonary oedema is particularly prone to occur during labour and during the first twelve hours following delivery. However he offers no satisfactory explanation why this should be the case. Again it would appear that the mechanics of the circulation are upset during and after labour, and that an increased amount of blood is suddenly returned to the heart causing it to fail. He also quotes Plasse (1936) who showed that there is some evidence that in rare cases labour may produce pulmonary oedema in patients who are not known to be suffering from any cardiac lesion, valvular or myocardial. The four deaths in this series from acute pulmonary oedema on whom post morten findings were negative, confirm this finding of Plasse. Jensen also states that acute pulmonary oedema is most common in young primiparous women. Of the small number in the series reported here all but one was under 30 years of age, but only two were primipara.

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There is no doubt that pulmonary oedema is the ultimate cause of death in many women who have congestive heart failure especially those dying a few hours after delivery. These cases are however included under deaths due to congestive heart failure.

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(c). BACTERIAL ENDOCARDITIS.

In this series 7 cases or 16 per cent of the total died as a result of bacterial infection of valves previously damaged by rheumatic endocarditis. 4 of these cases developed the infection in the course of puerperal fever, that is, they developed acute ulcerative endocarditis secondary to a septicaemia. One other case developed the infection during pregnancy and died 24 hours after a hysterotomy, the autopsy revealing acute ulcerative endocarditis (organism not known). From the history of this case it is not possible to tell if the infection on the valves was part and parcel of a general septicaemia or not, but probably it was.

Only 2 cases developed the subacute variety of bacterial endocarditis in the course of pregnancy. (It is possible that the infection was present before pregnancy occurred and that pregnancy aggravated the condition). It is impossible to tell whether pregnancy per se predisposes to bacterial infection of the valves, but it would appear that this is not

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the case although puerperal infection definitely does predispose to acute infection of the valves. The findings here do not agree with those of Bramwell (1948) who found that out of 22 women who had subacute bacterial endocarditis, 7 developed the infection during pregnancy. He had 2 deaths but concluded that if this complication is promptly diagnosed and adequately treated the prognosis is good for mother and child. He is of the opinion that pregnancy in itself is a predisposing factor in the development of subacute bacterial endocarditis.

There is little other information in the literature on the subject of subacute bacterial endocarditis associated with pregnancy and not many cases have been recorded. Jensen (1938) estimated the incidence to be about 1 per cent of all obstetrical cases with heart disease which is probably the same incidence as in the non-pregnant woman.

All the deaths recorded in this series occurred in pre-penicillin days, when development of bacterial endocarditis was invariably fatal. With new treatment of this complication it is possible that deaths due to this cause will not in future occur. Within the past year a pregnant woman who developed subacute bacterial endocarditis in the early months was successfully treated with penicillin and was delivered of a healthy live child.

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(d). EMBOLISM.

It is perhaps surprising that in this series no death occurred as a result of embolism either of cardiac or peripheral venous origin. Cardiac disease, especially when peripheral oedema is present, is a predisposing cause of phlebo-thrombosis in the legs with consequent risk of pulmonary embolism. There was however no fatal case of this type. In the absence of congestive failure there was no fatal case of pulmonary embolism with a cardiac origin of the embolus. It is true that pulmonary embolism was the terminal event in several cases with severe congestive failure, but it is considered that this was not the primary cause of death and these cases have therefore been listed under deaths due to congestive heart failure. Hamilton and Thomson (1941) in their series of pregnant cardiacs out of 54 deaths, had 8 due to embolism of cardiac origin and 3 due to pulmonary embolism of peripheral origin- a percentage of the total deaths of 14.8 and 5.5 respectively. In other words about 20 per cent of all the deaths were due to embolism. The reason for this high figure is probably due to their separating these cases from deaths due to congestive heart failure. In the present series such cases, if they had congestive failure, would have been included under this heading. It is considered that the

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majority of Hamilton and Thomson's cases did in fact have congestive failure.

One reason for the low incidence of embolism is the low incidence of cases of auricular fibrillation in the present series. Such cases were as a rule not allowed to go to term, but those that did, usually developed congestive failure which proved fatal. It would appear that embolism as a primary cause of death is not common during pregnancy but that it is a more common cause of death in older non-pregnant cases as the follow-up examination revealed 14 per cent of the total deaths due to this cause.

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(e). RHEUMATIC FEVER AND CHOREA.

In this series only one death or 2 per cent of the total occurred during an attack of acute rheumatic fever. In Hamilton and Thomson's series there were 2 deaths or 3.7 per cent of the total. It has been shown that acute rheumatic fever is not common in the adult and it appears that it is rarely associated with pregnancy. Several authors including Hamilton agree with this finding. However if the cases dying in congestive failure and pulmonary oedema are examined post mortem a surprisingly large percentage show evidence of recent acute rheumatic vegetations.

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In this series, of 20 autopsy examinations 11 or 55 per cent showed recent rheumatic activity. From these figures it is probably right to assume that roughly half of all cases going into congestive failure during pregnancy have active endocarditis in the absence of any clinical evidence of rheumatic fever such as joint and muscle pains. Typical acute rheumatic fever as it occurs in childhood appears to be very rare during pregnancy. Jensen(1938) states that acute endocarditis is an important cause of congestive heart failure during pregnancy and the findings in this series confirm this view. A further discussion on the subject will be given when the course of rheumatic heart disease is studied later.

These then are the main complications of rheumatic heart disease met with during pregnancy.

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## 6. MANAGEMENT OF CASES DURING PREGNANCY AND METHOD OF DELIVERY.

It is not within the scope of this thesis to discuss fully the management and treatment of cardiac cases but it can be pointed out that much improvement has taken place in the care of pregnant cardiac cases since over 70 years ago when MacDonald (1878) reported a 55 per cent mortality rate in such cases. Even in the past 20 years there has been a marked improvement

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in the mortality rate from roughly 7 to less than 2 per cent. Various factors combine to make this advance and these will be discussed briefly.

(a). SELECTION OF CASES.

From experience gained in the past and from the statistical evidence given in this paper we now know with almost complete certainty which women are going to do badly during pregnancy and consequently these women should not be allowed to become pregnant, or should they become pregnant the pregnancy should be terminated at an early stage. Therapeutic abortion in cardiac patients can only be done with safety before the end of the third month of gestation so that in order to select patients it is necessary that they be examined early in pregnancy - before the third month.

We know that all cases who are in functional grade IIb or III or have ever been in that grade previously will do badly. Also any case exhibiting auricular fibrillation (usually grade IIb or III anyhow) should not be allowed to proceed. The danger lies not in the arrhythmia per se but in the fact that such cases have advanced rheumatic heart disease. Also most cases with evidence of active rheumatic infection should have the pregnancy terminated. One of the factors in the lowering of the mortality rate has been the application of these

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rules to more and more cases in recent years, as more and more cases have been referred early to the cardiac clinic. As Haultain (1948) points out, during the war years pregnancy virtually became notifiable, in order that special rations etc., could be obtained. Because of this, adequate ante-natal supervision could be given with resultant reduction in maternal mortality and morbidity.

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(b). ANTE-NATAL SUPERVISION.

Having decided that pregnancy continue adequate ante-natal supervision is essential. The prevention of congestive heart failure is the main aim. This is gained by reducing all unnecessary work, ensuring adequate rest and sleep, treating anaemia and preventing the development of respiratory infections. By these means the majority of cases go through pregnancy with little difficulty. More prolonged rest may be necessary in some cases and complete bed rest required for cases that fall into grades IIb or III.

Better ante-natal supervision in an increasing percentage of cases (booked cases) has tended to reduce the maternal mortality.

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(c). TREATMENT AND PREVENTION OF THE COMPLICATIONS.

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With the exception of subacute bacterial endocarditis the treatment of the complications of cardiac disease during pregnancy has not altered to any extent during the past twenty years or so. No advance has been made in the therapy of congestive heart failure, the commonest complication. Fortunately with the aid of penicillin it is now possible to treat effectively the cases of bacterial endocarditis which were previously always fatal, thus eliminating a complication which accounted for 16 per cent of the fatal cases. The big reduction in the mortality rate has occurred with the prevention of the most common complication, congestive heart failure, by methods just given and not by any change in treatment. The emphasis is on prevention, not on treatment. Given proper facilities, fatal cases of congestive heart failure should not occur. Unfortunately the same cannot be said for the complication of acute pulmonary oedema which occurs without warning and may prove fatal in a very short time. It seems impossible to forestall this event and it can be expected that death from this cause may increase in importance.

Should congestive failure develop complete bed rest is essential with in addition digitalis and mercurial diuretics and even low salt diets in the more resistant cases. With this treatment the

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majority of cases graded as IIb rapidly revert to grade IIa. The grade III cases as a rule do well but such cases should be confined to bed for the remainder of pregnancy if a relapse is to be avoided. Too often such cases if allowed to go home, reappear a few weeks later in a more advanced stage of failure. Gorenberg (1943) advocates gomplete bed rest (until the end of pregnancy) for all cases showing a significant decrease in cardiac reserve and also increased bed rest for all cases in grades I and IIa in the last 3 months of pregnancy. By this means he reduced the incidence of congestive heart failure from 22 per cent to 0.5 per cent with consequent reduction in maternal mortality rate.

By these three means - selection of cases, ante-natal care and treatment of complications - it has been possible to reduce the mortality rate during pregnancy, mainly by eliminating bacterial endocarditis as a cause of death and decreasing the incidence of congestive failure which is by far the commonest cause of death. In future it should be possible to prevent deaths from these causes during pregnancy and a cardiac patient, having been allowed to embark on pregnancy, should be liable to only those risks likely to occur in a healthy pregnant woman, with one exception, namely the development of acute pulmonary oedema which is at present an unpredictable complicat: :ion.

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(d). OPERATIVE INTERFERENCE AND METHOD OF DELIVERY.

As has been pointed out, the main aim during pregnancy is the prevention of congestive heart failure, firstly as such a complication may prove fatal, and secondly as labour in the presence of congestive failure is a serious event. It has been shown that most of the deaths from this cause occur in the few hours following delivery and it is therefore imperative to have a cradiac patient in as good condition as possible for the added efforts of labour.

In Part I it was concluded from all the available evidence that the mortality rates following Caesarean Section were higher than following Pelvic delivery in women with congestive failure. It has also been the impression that operative interference such as a hysterotomy even in the early months in the presence of congestive failure is an undertaking of considerable risk to the patient. The reason why operative interference should be dangerous in the presence of the more serious grades of heart failure is not known for certain. Snyder (1938) studied the effects of surgical operations on the output of the heart in nine patients, and found a decreased output of 41 per cent immediately after operation, several days elapsing before the output returned to normal. The diminished output is presumably due to the

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circulatory readjustments coincident with a state of surgical shock which in some degree is almost inevitable following surgery. A reduction of 41 per cent in the output of a heart already in failure would be a serious occurrence and may partly explain why such cases do badly following operation. Other factors probably play a part. Firstly there is decreased diaphragmatic movement following abdominal operations with resultant inadequate ventilation and tendency to even further stasis in the already congested lungs so predisposing to infection. Secondly there is the added risk following inhalation anaesthesia in cases of congestive failure, though with modern anaesthesia this should not be of very great significance. Thirdly there is the increased risk of complications, such as sepsis and embolism, which may follow any operation and this risk is probably even further increased in patients with congestive failure.

None of these factors are applicable to cases delivered from below which would explain why clinically the strain following Caesarean Section is greater than after a natural vaginal delivery. The older view that Caesarean Section, as it did away with the strain of labour was found to be an advantage to the cardiac woman, has been steadily losing ground for many years. Even in 1938 Jensen stated that since 1900 the use of Caesarean Section had been greatly

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developed and in some countries overdeveloped but concluded that the trend in America was towards a more conservative attitude. In this country as early as 1931 McIlroy and Rendel (1931) were probably the first to realise that non-intervention was the better rule. Since then facts and impress: :ions have accummulated and the findings were summarised in Table XIII.

In this series, taking only patients in congestive failure, the death rate following Caesarean Section was 12.4 per cent compared with 6.8 per cent following Pelvic delivery. Medelson (1944) comparing a similar series of cases had a mortality rate of 9.5 per cent following Caesarean Section and no deaths following Pelvic delivery. Gorenberg and McGleary (1941) and Hamilton and Thomson (1941) produce somewhat similar figures without stating the actual grade of the patients concerned. Stromme and Kuder (1946) report an increased use of forceps deliveries with a reduction in use of Caesarean Section to just over one in a hundred cases.

While the evidence in all respects is that Caesarean Section in the presence of congestive failure is more dangerous than a pelvic delivery it is considered that one of the main reasons why operative interference is now less used is that patients are being presented at term in much better

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condition and seldom are severe cases of congestive failure met with. What used to be the main indication for Caesarean Section is therefore not present. Gorenberg (1943) gives a good example of this. He insisted on complete bed rest for the remainder of pregnancy for all cases who became grade IIb or III and increased rest in all other cases. He thereby decreased the incidence of congestive heart failure at term from 22 per cent down to 0.5 per cent. Surgical intervention was not permitted and labour was allowed to occur spontaneously. As a result the death rate in cardiac cases fell from 3.5 to 0.6 per cent. It is probable that the better ante-natal care, rather than the absence of operative interfer: :ence, was the cause of the reduced mortality rate, in his series of cases. Bunim and Rubricius (1948) state that the presence of rheumatic heart disease, per se, is no longer acceptable as an indication for Caesarean Section but add that it may occasionally be resorted to as a means of terminating a prolonged labour in order to reduce the danger of heart failure. There is little doubt that in well compensated cardiacs operative interference is tolerated as well as in normal individuals and there should be no fear in performing a Caesarean Section on such a case where the indication is an obstetrical one.

To sum up it can be stated that operative

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interference in the presence of heart failure is more dangerous than a pelvic delivery, and the decreased use of Caesarean Section on such cases has probably brought about a reduction in maternal mortality rate following delivery but it is considered that the rate has been reduced even more by better ante-natal care and selection, so that patients are brought to term well compensated and with no evidence of failure.

What has been said of operative interference at term applies equally at any time during pregnancy. A hysterotomy to terminate pregnancy in a Grade III patient say at the fifth month is a dangerous procedure and should not be attempted. It is for this reason that early examination of cases is essential. Before the third month it is relatively easy to terminate pregnancy from below without undue upset to the patient. Thereafter operative interference is necessary. In spite of Gorenberg's claim to have reduced the incidence of congestive heart failure to negligible proportions, cases are still encountered which in spite of the most energetic medical treatment, show persisting signs of congestive failure. Such cases are bad risks, especially if the period of gestation is advanced far enough to prevent therapeutic vaginal abortion. There is a tendency to be forced into operative interference, but from available information and impressions, this is a step very likely to prove

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fatal. It must be remembered that during the final month of pregnancy there is an appreciable drop in the cardiac output and clinical improvement can be expected. It is considered far less dangerous to allow the pregnancy to proceed in such conditions, than to terminate by operation. Again if such cases were seen early the difficulty would not arise.

To sum up this section on the management and method of delivery of cardiac cases it can be stated that early selection and better ante-natal care has caused a marked reduction in the maternal mortality rate by preventing death from the commonest complication - congestive heart failure - both during pregnancy and following labour. The policy of non-intervention in cases of congestive failure is also considered to have reduced the mortality rate to some extent, and finally the effective treatment of subacute bacterial endocarditis has almost eliminated this complication as a cause of death.

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## 7. THE EFFECTS OF PREGNANCY ON THE NATURAL COURSE OF RHEUMATIC HEART DISEASE.

To understand fully the effect of pregnancy on the course of rheumatic heart disease it is necessary to know something about the natural history of the disease. This has been studied carefully by Cohn and Lingg (1943a & b) and DeGraff and Lingg (1935)

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and these workers divided the course into four phases:

(1) The initial infection - acute rheumatic fever, chorea, carditis and vague muscle and joint pains.

(2) One or more recrudescences of the initial infection.

(3) The inactive period with no evidence of rheumatic activity and evidence of only slight decrease in functional capacity.

(4) A period with more or less rapid diminution of cardiac reserve leading to congestive failure and death.

In the series of 295 cases studied it was found that 43 per cent of the cases giving a rheumatic history had no reinfection and therefore did not pass through phase 2. In those cases phase 1 usually had a duration of under one year. In the remainder the second phase lasted anything between 2 and 10 years. One quarter of all the cases gave no clinical history of having passed through either phase 1 or 2. It appears then that rheumatic carditis can occur in an apparently healthy individual. Since this is the case it is reasonable to assume that reactivation of carditis can occur in known subjects of rheumatic heart disease without any clinical evidence of the activity. This is an important point because it may explain why some women go into congestive failure at a much earlier age than others. In this series

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the peak incidence of the rheumatic infection was at 12 years of age and half of all the cases received their first infection between the ages of 8 and 14. In Cohn and Lingg's (1943a) series the maximum incidence was at 8 years, but they were dealing with all cases and not just those who had reached the childbearing period. Rheumatic infection for the first time is very rare after the age of 20. The average duration of phase 3 - the inactive phase - can be calculated by taking the average age of patients in phase 1 (12 years), and adding 5 years as the mean duration of phase 2, giving a figure of 17 years as the average age when rheumatic activity has subsided, and subtracting this figure from the average age of patients in grade IIa - in this series 35 years - giving a duration of 18 years in phase 3. The final phase usually occupies another 5 or 6 years.

From the detailed study of the rheumatic infection given in Part II it is apparent that the rheumatic state may show itself in various ways. Firstly, there may be muscle and joint pains of the acute variety as in typical rheumatic fever or of the subacute variety as in growing pains. Secondly, there may be evidence of chorea and thirdly there may be carditis either obvious clinically in its initial stages (e.g., pericarditis) or more often completely occult clinically, evidence of previous carditis being discovered years later in the form of valvular damage. Any of these manifestations of

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the rheumatic state may occur singly or may be associated with each other, either simultaneously or consecutively the commonest being the association of carditis and acute rheumatic fever and the rarest being the association of chorea and acute rheumatic fever. A very large percentage of patients who develop the rheumatic state give a history of upper respiratory infection and presumably the disease is due to an altered response on the part of the patient to the streptococcus. Whatever the cause, once cardiac damage is sustained, a period of only about twenty years of active life can be expected after which a few years of increasing incapacity leads to death most commonly from congestive heart failure. During the final years auricular fibrillation commonly develops and this arrhythmia is an indication of the advanced state of the disease.

The course of the disease may be altered at any time, firstly, by the development of active carditis with or without evidence of rheumatic fever, secondly by subacute bacterial infection of the valves and thirdly, occasionally, by embolic phenomena in the presence of normal rhythm. The development of active carditis appears to be relatively common and it is suggested that this is the cause of congestive failure in younger women with normal rhythm of the heart. Active carditis was found at autopsy in 55 per cent of cases dying during pregnancy in congestive

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failure. It appears therefore that active carditis is relatively common in adults of the childbearing age but that the other manifestations of the rheumatic states are very uncommon. Acute rheumatic fever and chorea are rarities in adult life. The recognition of active rheumatic carditis is very difficult clinically. It can be suspected and is very likely present in cases of congestive failure in younger subjects where normal rhythm is present. Provided the course of the disease is not altered death usually takes place from congestive heart failure or from embolism, either pulmonary or peripheral, and the average age at death is around 40 years in those who reach adolescence.

Childbearing produces the greatest physiological burden on the heart and it is not surprising therefore that in patients with little or no cardiac reserve, pregnancy will probably prove fatal. On the other hand the vast majority of women with heart disease nowadays successfully overcome pregnancy and labour but it is not known for certain if the course of the disease is affected by pregnancy or repeated pregnancies. The study of this series of cases has shown that pregnancy may induce temporary congestive heart failure in a woman roughly 5 to 7 years before it appears again in the normal course of events. We do not know however whether damage is sustained during pregnancy from which a

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patient never really recovers, or in other words, whether pregnancy has an adverse effect on the course of the disease. Up till recently it has been the view that pregnancy takes something from the cardiac woman's expectancy of life.

Heaney (1922) stated "that many women with valvular disease of the heart who might have lived as spinsters to a ripe old age, have had their lives shortened, have become prematurely invalided or have died because they have become mothers, is known to any physician of experience." All other authors around this time confirmed this view. Even in 1937 Lamb (1937) stated that post partum 10 out of 23 of his cases were worse. As Jensen (1938) points out supporters of this view seem to consider cardiac reserve as a fixed amount, like money in the bank, subject to withdrawal at demand and that pregnancy involves an excess withdrawal of this reserve and thereby hastens its complete exhaustion.

The more modern view, first expressed as long ago as 1917 (Gellhorn, 1917) is that pregnancy in itself does not shorten life in a woman with valvular disease of the heart. There are great difficulties in attempting to prove or disprove this latter view. It is necessary to compare parous with nulliparous women in order to observe the effects of pregnancy. If this is done it is found that the nulliparous group is weighted by women too ill to marry or to

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have children. Males cannot strictly speaking be used for comparison as then the factor of sex might interfere. Nevertheless many interesting studies have been made using the age at death of nulliparous and parous women as a comparison. Gilchrist and Murray-Lyon (1933) found no significant difference in the age at death in the two groups. Jensen (1938) summarising the work of various authors, found that death occurred earlier in nulliparous as compared with parous women. As has been pointed out the reason for this finding is that the nulliparous group is probably weighted with women too ill to have children. However as Boyer and Nadas (1944) have pointed out if figures are taken which include only women surviving to a marriageable age and only those dying in congestive heart failure there was no significant difference in the average age at death between nulliparous and parous women.

A similar study was here made between parous and nulliparous women and also men but instead of using the age at death as a basis for comparison the age of development of severe congestive failure (grade III) was used. Patients who had been admitted to the general medical wards of the Royal Infirmary of Edinburgh in congestive heart failure were the subject for study. No case under the age of 18 was included. The findings are given in Table XXXV. It will be seen that the average age

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of onset of severe congestive failure in 87 parous women was 40.6 years, in 65 nulliparous women 42.3 years and in 71 men 41.8 years.

TABLE XXXV.

Average Age of Occurrence of Congestive Heart Failure of Rheumatic Origin in different Groups of Patients from a General Hospital.

| | Number. | Average Age. |
|---|---|---|
| Men. | 71 | 41.8 |
| Nulliparous Women. | 65 | 42.3 |
| Parous Women. | 87 | 40.6 |

In addition 12 cases who on follow-up examination were in severe congestive failure had an average age of 41.0 years (Table XXIV). These figures indicate that there is no significant difference in the course of the disease as far as the onset of congestive failure, between men, parous and nulliparous women.

To summarise the findings then it appears that there is no appreciable difference in the course of the disease in parous as compared with nulliparous women using the age of onset of failure and the age at death as bases for comparison. Some criticism has been levelled at such methods of comparison and it is stated by some authors (Bunim and Rubricius, 1948).....

1948) that several important factors in the nulliparous and parous groups have not been controlled. These include age at onset of heart disease, number and severity of recurrences of rheumatic fever and the respective duration from onset of heart disease to failure and from failure to death. As has been pointed out in this thesis before, the age of onset of heart disease is very difficult to determine and consequently the duration of heart disease can only be known in roughly three quarters of the cases. Also the findings in this paper indicate that the severity and number of recurrences of attacks of rheumatic fever bear no relationship to the ultimate fate of the patient who has overcome these acute attacks. Anyhow these variable factors mentioned by Bunim and Rubricius are probably equally distributed in both groups (parous and nulliparous) provided a sufficient number of cases is used so that really the absolute age at onset of congestive failure and absolute age at death can be true bases for comparison in the two groups.

Nevertheless, Cohn and Lingg (1949) in assessing the effects of pregnancy on the course of heart disease took several factors into account. They selected patients both parous and nulliparous from a large group admitted over a period of years to selected cardiac clinics affiliated with the New York

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Heart Association, who had been followed through many years until death. As a result, out of 1,563 such patients (females) they found that of those who lived into the childbearing age 567 had and 314 had not borne children. Of the parous group only 169 were included in their study since it was necessary to limit the analysis to those cases for which medical records, including the examination of the heart at the time of parturition, were available. Similarly, 99 of the 314 nulliparous cases were excluded, either because the disease began after, or late in, the childbearing period, or because before that age the course of the disease had been more severe as indicated by factors such as auricular fibrillation, and prognosis might therefore be expected to be less favourable than in the parous group. In order to compare the two groups it was necessary to be reasonably certain that the nulliparous group was not weighted by women too sick to bear children. This turned out to be their most difficult problem. They divided the two groups, parous and nulliparous, into three Classes. Class I consisted of patients in whom the disease was advanced or moderately advanced at the beginning of the childbearing period. Class II consisted of patients in whom the disease was less advanced at the beginning of the childbearing period and Class III of patients in whom the disease began after adolescence or in the childbearing period of life.

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In attempting to judge the effect of parity on the course of the disease they compared the two groups, parous and nulliparous, with respect to (a) symptoms and signs of cardiac insufficiency, (b) the development and extent of valvular lesions,(c) episodes of congestive heart failure, (d) the incidence of auricular fibrillation and (e) the duration of the disease. Having made this comparison in each of their three Classes, there was no significant difference between the parous and nulliparous groups. It is perhaps interesting to note that auricular fibrillation complicated the disease in about 45% of all cases. They state that there was no appreciable difference in the curves of duration of disease or age at death in the parous and nulliparous groups, and they concluded that there was no reason for believing, other things being equal, that childbearing affects adversely the course of rheumatic heart disease. They considered, however, that the number of cases (384) was too small for reliability. Nevertheless this is the largest number of cases that has been studied in such detail.

It has been argued, that if pregnancy had an adverse effect on the course of the disease, repeated pregnancies would have an even worse effect. In the series studied in this thesis, it was found that deaths occurred, within a given period, in the same proportion in women who had one, two or multiple

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pregnancies. The statistical analysis indicated that the number of pregnancies bore no relation to the prognosis. Also by using the functional grade observed during and after pregnancy as a basis for comparison it was found that the course of the disease as estimated by the downward trend in grade over the years was not affected by the number of pregnancies, the curve being the same in each of the groups.

Such a comparison between women with one, two or multiple pregnancies is however not strictly fair. We are dealing here with a specially controlled group of patients, in that the number of pregnancies has been limited in some and not in others. Nevertheless the findings seem to indicate that the number of pregnancies per se has no effect on the ultimate fate of women with rheumatic heart disease. There is no doubt that if cardiac women were allowed to have repeated uncontrolled pregnancies, death would eventually occur during a pregnancy and multiparous women would therefore die at an earlier age than women who had borne just one or two children. However in a controlled group such as this, where repeated pregnancies are only allowed under certain rules, the number of pregnancies per se appears to have no effect on the ultimate course of the disease. One must remember Gilchrist's (Gilchrist and Murray--Lyon, 1933) criticism that only women in good health are allowed to have many children.

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All the available evidence goes to show that pregnancy does not alter the course of rheumatic heart disease. Pregnancy will however impose an additional burden on the heart which may in certain cases prove overwhelming but provided the pregnancy is safely overcome the expectation of life in any woman is not shortened. Pregnancy can cause complications which may prove fatal and the causes of death during pregnancy occur in roughly the same proportion as the causes of death in the non-pregnant state, with one exception, namely that embolic deaths are uncommon during pregnancy but fairly common in women who have survived pregnancy. This is due to the fact that the majority of embolic deaths are associated with auricular fibrillation which arrhythmia is seldom encountered during pregnancy. The Registrar General for Scotland has kindly supplied the figures for the death rate due to rheumatic heart conditions in males and females between the ages of 20 and 40. The figures are given in Table XXXVI.

It has not been possible to obtain the incidence of rheumatic heart disease in the general population between the ages of 20 and 40 but taking the death rate as a guide it would appear that rheumatic heart disease is roughly twice as common in women as in men. The figures given earlier in the paper show that about 1 per cent of women who

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become pregnant have heart disease, in 95 per cent of cases of rheumatic origin.

TABLE XXXVI.

Death Rate per 100,000 at Ages 20-39 in Cases with Rheumatic Heart Disease.

| Scotland. | Males. | Females. |
|---|---|---|
| 1941 | 6 | 18 |
| 1942 | 10 | 17 |
| 1943 | 8 | 18 |
| 1944 | 9 | 15 |
| 1945 | 9 | 13 |
| 1946 | 10 | 15 |
| 1947 | 8 | 16 |
| Average | 8.5 | 15.3 |
| Edinburgh. | | |
| 1941 | 10 | 14 |
| 1942 | 6 | 17 |
| 1943 | 5 | 19 |
| 1944 | 15 | 11 |
| 1945 | 10 | 14 |
| 1946 | 8 | 14 |
| 1947 | 7 | 9 |
| Average | 8.5 | 14 |

It can therefore be said that the incidence of rheumatic heart disease amongst all women of the childbearing age (20-40) is 1 per cent approximately. The death rates in Table XXXVI are given for 100,000 of the healthy population. 1 per cent of this figure (100,000) is 1,000 so that the death rate in rheumatic heart disease from all causes (including pregnancy) is 15.3 per 1,000 for Scotland and 14.0 for Edinburgh, or roughly 1.45 per cent in the

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childbearing age. The death rate during pregnancy in cardiac cases has dropped to between 1 and 2 per cent in recent years, so that it can be said that there is little if any additional risk to life during pregnancy as compared with the non-pregnant state, provided the modern rules for selection and treatment are enforced. With this knowledge and with the knowledge that ultimately pregnancy does not shorten life we are justified in continuing to use the present method of selection and management of cardiac cases.

The practical application of the findings in this thesis are firstly that a cardiac woman should have her children at an early age, preferably in the early twenties before there is increased risk of decompensation: secondly that the number of children she hears will not adversely affect her ultimate health provided the functional capacity of her heart is good during and between pregnancies: and thirdly that functional grading is the most important method of assessing prognosis both during and after pregnancy.

## SUMMARY.

A review of the work of the past twenty years in the Cardiac Clinic attached to the Royal Simpson Maternity Pavilion of the Royal Infirmary of Edinburgh and a follow-up examination of all cases that had attended this Clinic in the second ten-year period, were carried out. The incidence of organic heart disease was determined to be about 1 per cent of all pregnancies and the mortality rate in the last few years to be just under 2 per cent. Considerable reduction in the death rate had occurred over the period of years and the reasons for this were discussed. One of the main factors was considered to be a reduction in the number of cases delivered by Caesarean Section and it was shown that the mortality rate following Caesarean Section was roughly twice that following Pelvic delivery in similar groups of cases.

The complications and causes of death in cardiac cases during pregnancy were analysed and discussed. The most important single factor was the development of congestive heart failure and means of reducing the incidence of this and other complications were discussed. The majority of deaths occurred in the puerperium usually within one day of delivery and it was concluded that the

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mortality rate had been reduced and further reduction could be possible by better and earlier selection of cases as to their fitness to proceed with pregnancy, better ante-natal care with the use of absolute bed-rest when necessary, the prevention and early treatment of the complications liable to occur during pregnancy and a policy of "non-intervention" in cases showing, or having shown, congestive heart failure. It was concluded, on all the available evidence, that Caesarean Section was not the method of choice and was not indicated in cardiac patients unless there was some other obstetrical reason for carrying it out.

On re-examination of the cases it was found that 10 per cent of those regarded as cardiacs during pregnancy had in fact no organic heart disease. These cases were discussed in detail and it was concluded that in certain women, for some reason as yet unknown, apparent cardiac failure can occur during pregnancy. The most likely explanation was residual myocardial damage of rheumatic origin, but no definite proof could be given. Acute sudden failure may also occur in women with normal hearts during or immediately after labour and the reasons for this were discussed.

Of those who had definite organic heart disease during pregnancy 95 per cent had rheumatic heart disease the next most important group being composed of congenital lesions. The difficulty in

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diagnosing organic heart disease during pregnancy was discussed and it was concluded that in some cases it was impossible to decide with certainty if an organic lesion were present or not. The main clinical findings that gave rise to a wrong diagnosis having been made, were listed and discussed.

It was found that the functional capacity of the heart, as determined by the method of grading which has been used for over twenty years, was the most important factor in assessing prognosis during and after pregnancy. The relationship between the functional capacity and several other factors was carefully studied and it was concluded that a woman's grade bore no relationship to her past rheumatic history or the type of valvular lesion but did bear some relationship to her age.

In assessing prognosis in a pregnancy knowledge of functional capacity in a previous pregnancy gave full information, and Tables of prognosis based on age, and grade in a previous pregnancy were compiled.

As to the ultimate effects of childbearing on the life of a woman with heart disease, it was concluded that, provided the pregnancy and puerperium were safely overcome, she had sustained no permanent irreparable damage. Pregnancy itself might induce congestive heart failure roughly 5 to 7 years before

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it was due to appear in the normal course of events but with modern selection and ante-natal care it was doubtful if there was really much of an increased risk attributable to pregnancy. Provided the heart remained well compensated the number of pregnancies did not affect the prognosis.

It was possible from the statistical analysis to justify the practice of preventing women who had decompensated at any time from having another pregnancy, and it was suggested that care should be taken in allowing women in their late thirties or early forties to continue with pregnancy even although they were fairly well compensated.

It was concluded therefore that the present methods of dealing with cardiac cases were justifiable and provided these methods were carried out pregnancy would not adversely affect the course of heart disease.

In practice it was considered that if a choice can be made, a woman with heart disease should have her children at an early age and have fairly short intervals between pregnancies, and even though the number of pregnancies does not affect the prognosis, it is probably best to limit the family to two or three children.

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